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INDIVIDUAL DIFFERENCES IN SUSCEPTIBILITY TO INDUCED DEPRESSED
MOOD

by

Laura Garcia-Browning

A Thesis
Submitted to the Faculty of Graduate Studies and Research
Through the Department of Psychology
In Partial Fulfillment of the Requirements for
The Degree of Master of Arts at the
University of Windsor

Windsor, Ontario, Canada

2005

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395 Wellington Street
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ISBN: 0-494-09738-8

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ISBN: 0-494-09738-8

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Abstract

One hundred and seventy-eight men and women were exposed to the negative Velten mood induction procedure in order to examine how individual personality characteristics contributed to differences in their responses. Personality characteristics measured were chosen based on relationships with naturally occurring depression (locus of control, emotional intelligence, neuroticism, negative attributional style, self-critical perfectionism, rumination, silencing the self score). Locus of control and silencing the self score was correlated with change in happy mood and emotional intelligence was correlated with and predicted change in happy mood, while neuroticism was correlated with and predicted change in sad mood. Differences in findings related to happy and sad mood scores and the influence of emotion regulation on the results are discussed.

Acknowledgements

I would like to thank Dr. Jim Porter for being an incredibly dedicated and understanding supervisor. I would also like to thank the members of my committee, Dr. Carty and Dr. Hibbard for their invaluable time and input.

Mom, Doug, Alex, Dad, LeeAnne, Lauri- I could not have done it without you. Thank you for everything.

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Chapter 1

INTRODUCTION

*Overview**Context of the Problem*

Clinical unipolar depression is an intensely debilitating disorder that affects approximately three to four percent of Canadians each year (Patten & Lee, 2004). Research has indicated that unipolar depression has the potential to be under-diagnosed (Pomerantz, 2003), particularly among psychiatric patients (Macaskill & Macaskill, 1999) and the elderly (Harpole & Williams, 2004; Goodwin, & Smyer, 1999). This suggests that rates of clinical unipolar depression may be higher than estimated. According to the Diagnostic and Statistical Manual (4th Edition, American Psychiatric Association, 1994) clinical depression can only be diagnosed when either anhedonia and/or depressed mood is present for a period of at least two weeks. As such, depressed mood is a central component of clinical depression because, “Sad moods are the most prominent and pervasive emotional symptoms in depression” (Gillig & Gillig, 1995, p. 3). People suffering from clinical depression must deal with devastating emotional and physical symptoms, yet analysis of psychotherapy treatments for unipolar depression report an average success rate for only 70% of clients (Lambert & Bergin, 1993). In addition, what is considered the most efficacious treatment of clinical depression, a combination of psychotherapy and medication, boasts success rates of only 72-73% (Otto, Smits, & Reese, 2005). A more thorough theoretical understanding of this mental disorder is required in order to fuel more potentially successful treatments.

In addition to the impact of clinical depression, mild to moderate forms of depression also drain community resources and impair people's ability to enjoy life (Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Research by Johnson, Weissman and Klerman (1992) demonstrated that "physicians provided more services on average to those with 'depressive symptoms' than those with 'depressive disorders', establishing that significant morbidity is associated with subclinical depression" (as cited in Parker, Wilhelm & Asghari, 1998, p. 10). According to Seligman, "mild depression, in and of itself is an enormously widespread and significant problem, its cost in misery and loss of productivity is untold, and I find it callous that investigations of the cause and cure of mild depression should be dismissed as analogues to some other, more real problem." (1978, p. 177). As such, the entire range of depressive symptoms, from mild subclinical levels of depression to severe clinical depression, is worthy of study. The literature indicates that mood induction techniques can provide a method of examining the factors that cause vulnerability to depression and depressed mood (Martin, 1990).

Definition of Terms

For the purposes of this discussion, the following terms will be employed. 'Depressed mood' will refer specifically to "a pervasive and sustained emotion that colors the perception of the world" (DSM-IV, 1994, p. 825), in other words the naturally occurring (non-induced) emotional aspects of a depressed state. 'Induced depression' will refer specifically to mood states resulting from exposure to mood induction procedures. 'Naturally occurring depression' will refer to naturally occurring (not induced in the laboratory by mood induction techniques) depression, and will include not only the associated mood states but also other symptoms. At times, naturally occurring depression

will be further sub categorized based on levels of symptom severity and duration:

‘clinical depression; meaning that symptoms have met DSM-IV criteria for the diagnosis of depression; and ‘subclinical’ or ‘mild depression’ meaning that symptoms have not met DSM-IV criteria for diagnosis of depression.

Organization of Review

In the following sections, the similarities and differences between depressed mood and naturally occurring depression will be explored. In addition, the benefits of using mood induction procedures to investigate naturally occurring depression vulnerabilities will be discussed. A description of a variety of mood induction procedures and their utility as compared to the Velten Mood Induction Procedure (VMIP; Velten, 1968) will be followed by specific information regarding the theoretical background of the VMIP, as well as the validity of the VMIP. Naturally occurring mild and severe depressed mood states and the mood states induced by the VMIP will be compared and contrasted. Finally, problems with the VMIP will be discussed, with specific attention to individual variability in the efficacy of the VMIP. Research examining individual differences in susceptibility to the VMIP will be reviewed in order to explore potential factors which may contribute to vulnerability to depressed mood states.

Empirical Review

Depressed Mood vs. Clinical Depression

How can examining the effects of depressed mood states help us better understand the more complex and enduring phenomenon of clinical depression? An examination of the similarities and differences between these two will allow an understanding of what depressed mood can and cannot tell us about clinical depression. Parker, Wilhelm and

Asghari (1998) addressed this issue by examining if the quality and quantity of depressed mood states differed between participants with and without clinical depression. Parker et al. defined a depressed mood using a previously validated definition: “a significant lowering of mood, with or without feelings of guilt, hopelessness or helplessness, or a drop in one’s self esteem” (p. 11). Using a longitudinal design spanning 15 years (beginning in 1978, with assessment of depressive mood episodes occurring in 1978, 1983, 1988, and 1993), Parker et al. compared the characteristics of ‘depressed mood’ in 156 participants, 35% of whom were eventually diagnosed with Major Depression, 22% of whom met criteria for minor depression and the remainder of whom were not classified with any depressive disorder. Ninety-six percent in 1978, 99% in 1983, 97% in 1988 and 98% in 1993 of their participants endorsed experiencing at least one episode of a depressed mood in the 12 month period before each assessment. According to Parker et al.:

“Depression can be conceptualized as a mood state, a syndrome or a disorder. A depressed mood is generally viewed as a relatively ubiquitous phenomenon of trivial or modest severity, and thus distinct from the formalized depressive disorders, with their imposed severity and duration criteria, which are experienced by only a minority of people over their lifetime. ...[It] has been quite reasonably assumed that ‘major depression’ must therefore comprise more severe expressions of depression. ... An important additional parameter is duration, with recent DSM systems imposing a minimum duration of 2 weeks for ‘major depression’, so recognising that depression (or at least depressed mood) is commonly transient.” (Parker, Wilhelm, Asghari, 1998, pp. 10-11)

By comparing the quality and quantity of the depressive episodes experienced by people with and without clinical depression, Parker et al. were able to pinpoint the similarities and differences between a depressed mood in an

individual who has never been diagnosed with clinical depression and a depressed mood in an individual who has. They found the following quantitative differences between the experience of naturally occurring depressed mood for clinically depressed vs. non-clinically depressed participants:

1. Length of episode: people with Major Depression had longer episodes than people with minor depression who had longer episodes than participants with no diagnosis of depression.
2. Number of symptoms: people with Major Depression had more symptoms per depressive episode than people with minor depression who had more symptoms per episode than participants with no diagnosis of depression.
3. Frequency of episodes: people with Major Depression had more episodes than people with no diagnosis of depression. There were no significant differences between the frequency of episodes experienced by people with minor depression and those not diagnosed with depression.

Parker et al. also found qualitative differences between depressed mood experienced by people who were clinically depressed, mildly depressed, and people with no experience of depressed mood. Participants diagnosed with Major Depression experienced more intense tiredness, sense of despair, pessimism about the future, loss of pleasure, and appetite loss during their depressed mood than participants not diagnosed with depression. “Such analyses indicate that depressed mood states in those who have experienced a lifetime major depressive episode are distinguished by a higher rate of representative depressive mood and syndromal features.” (p.12). There were no

significant differences in quality of symptoms experienced by participants diagnosed with minor depression as compared to people not diagnosed with depression.

These results indicate differences in symptom degree but not in kind between depressed mood states experienced by individuals with and without a history of clinical depression. These results strongly support the contention that the differences between depressed mood states for individuals with Major Depression vs. individuals with minor or no depression are a matter of degree and not kind. This argument is termed the continuity theory of depression. Proponents of this theory “maintain that there is a linear association between mild, moderate, and severe forms of depression. In contrast, proponents of the qualitative view maintain that milder forms of depression are limited to a dysphoric mood state, whereas clinical depression is a distinct entity comprised of a constellation of symptoms, including sad affect, anhedonia, changes in sleep and eating behaviors, suicidal tendencies, and a profound sense of hopelessness and worthlessness.” (Flett, Vredenburg & Krames, 1997, p. 395). If depressive symptoms can be considered on a continuum, then the study of subclinical depressed mood will be informative with respect to more severe depressed mood states. Lewinsohn, Solomon, Seeley, and Zeiss (2000) looked at longitudinal data from large community samples of adolescents, adults and older adults. Their results also support a continuity view of depression symptoms, indicating a positive correlation between increasing levels of depressive symptoms and increasing levels of psychosocial dysfunction and substance abuse disorders. Lewinsohn et al. concluded that “the clinical significance of depressive symptoms does not depend on crossing the major depressive diagnostic threshold” (p. 345). Recently, an analysis of the personality characteristics associated with a number of clinical disorders at various

levels of severity revealed a systematic relationship between these traits and disorders, leading the authors to conclude that a “fundamental continuity [exists] between normal and abnormal psychological processes” (Watson, Gamez & Simms, 2005, p. 46). A recent review article of the continuity theory of depression, while conceding that there are still issues that need to be resolved, concluded that “most of the evidence at this time appears to favour both a manifest and latent continuum of unipolar depression symptomatology” (Solomon, Haaga & Arnow, 2001, p. 498).

In conclusion, there are two valid reasons for examining mild to moderate depressed mood states. The first is that these mood states, in and of themselves, are of clinical interest because they result in impaired quality of life and drain community resources (Lewinsohn, Solomon, Seeley, & Zeiss, 2000; Johnson, Wissman, & Klderman, 1992; Seligman, 1978). Secondly, as outlined above, mild to moderate depressed mood states provide information which can be extended to the understanding of the mood states associated with clinical depression.

Why Use Mood Induction to Look at Depressed Mood States?

A variety of procedures have been designed to induce temporary mood states analogous to naturally occurring moods in the laboratory. These mood induction tasks “may assist in the identification of those individuals who are at high risk of clinical mood disorders. For example, those individuals who are more vulnerable to emotional disorders following severe life events may also prove to be those who are more susceptible to the effects of particular mood induction procedures in the laboratory.” (Martin, 1990, p. 691). Similarities between the depressed mood states experienced by individuals with clinical depression and the depressed mood states experienced by individuals who do not have

clinical depression support the assumption that induced depressed moods are a useful analogue of clinically depressed mood states (Parker, Wilhelm & Asghari, 1998; Flett, Vredenburg & Krames, 1997; Watson, Gamez & Simms, 2005; Solomon, Haaga & Arnow, 2001; Lewinsohn, Solomon, Seeley, & Zeiss, 2000). As will be described in a following section, research indicates that there are enough similarities between induced depressed moods and naturally occurring depressed moods (at both subclinical and clinical levels) to utilize induced mood as a useful analogue tool to clinical depression.

Though the capacity of mood induction tasks to be used as tools in the study of depressive vulnerability has been identified in the literature (Martin, 1990; Clark, 1983), very few studies have examined the individual characteristics which make people more susceptible to induced mood. To date, no study has used this methodology to examine a large number of potential predisposing factors using a large sample of participants in order to determine how various individual differences contribute to depressive vulnerability. Such a study could indicate not only potential factors which could make an individual more susceptible to depressed mood, but also protective factors which may make a person more likely to feel positive emotions. As well, these factors may indicate potential treatment variables which could be altered in order to decrease depressive vulnerability. In addition, the use of regression analyses could inform the literature as to the relative contributions of a variety of individual characteristics to vulnerability to the experience of a depressed mood, information which has been lacking with respect of susceptibility to induced depressed mood.

A Brief Review of Various Mood Induction Procedures (MIPs)

Researchers have devised a variety of techniques for eliciting mood changes in the laboratory. Procedures have been developed to elicit a number of specific mood states such as depression, elation, anger and anxiety. The most widely used techniques include viewing a film, listening to music, exposure to social interactions and reading self-referential statements.

The film-viewing technique asks participants to watch mood related films. Some experimenters couple the film viewing with explicit instructions for the participants to attempt to experience the feelings suggested by the film. Films have been developed in order to reliably and strongly elicit sad emotions (Gross & Levenson, 1995; Philippot, 1993) but response to these films varies among individuals. In addition, some researchers use excerpts from popular movies to induce sad moods. Isen and Gorgoglione (1983) used clips from a movie entitled “Run” which depicts a man running from an unseen menace and then falling into his grave. Isen and Gorgoglione’s results indicate that negative moods induced using the film “Run” may last longer than moods induced by reading self-referential statements. More recently, Cavallo and Pinto (2001) induced depressed mood by asking participants to view an 11 minute film clip concerning domestic abuse, where a police officer watches a husband beat his wife without intervening. Westerman, Spies, Stahl and Hesse (1995) conducted a meta-analysis to compare the effects of a variety of mood induction procedures. Their results indicate that viewing of sad films coupled with explicit instructions to feel the desired mood was the most effective depressed mood induction procedure (mean effect size was $r_m=0.74$). Without explicit instructions, the film MIP had an effect size of $r_m=0.49$ (Westerman et

al., 1996). Unfortunately, the lack of a standardized film with which to induce depressed mood raises questions about the reliability and comparability of moods induced using this method.

The Music mood induction procedure was developed by Sutherland, Newman and Rachman in 1982. The Music MIP asks participants to listen to classical or modern music, and (as with the Film MIP) participants may or may not also be explicitly instructed to try and feel the desired mood state. Specific music used to induce depressed moods range from Albinoni's "Adagio in G minor" (Barnes-Holmes et al., 2004; Sutherland, Newman & Rachman, 1982) to Prince's "Sometimes It Snows In April" (Lewis, Dember, Schefft, & Radenhausen, 1995). The Music MIP has been found to be effective in inducing desired mood states. Westerman et al. (1996) found that the Music MIP had an effect size of $r_m=0.503$ with explicit instructions and $r_m=0.410$ without instructions. The Music MIP has also been found to induce higher mean levels of depression than control conditions (Clark, 1983). In addition, when participants are asked in post-experimental questionnaires whether they felt their mood change had been genuine, 87% of participants reported that it had been (Clark, 1983). In order to better understand how the music MIP induces mood change, Clark (1983) described the results of asking participants to explain the strategies they used to get into the desired depressed mood state. A variety of methods were reported including concentrating on the music, recalling past unhappy experiences, sighing, imagining possible future unhappy experiences, intensifying an already existing headache, and curling up into a ball. In addition, the Music MIP affects a large number of people. Sutherland, Newman, and Rachman (1982) found that 100% of participants met predetermined mood change criteria

when given the Musical MIP. This 100% success rate for the Music MIP has not been replicated; in fact Allwood, Granhag and Jonsson (2002) combined a Music MIP with viewing a film and reading a sad story and found that participants in the sad Music MIP condition did not differ on their pleasantness ratings than participants in the happy Music MIP. In other words, the sad MIP did not produce a change in the desired mood state direction. Martin's (1990) review of MIPs indicated a more encouraging success rate of 75% of participants experiencing the desired mood state criteria following a Music MIP. Critics of the Music MIP have suggested that some individuals can have idiosyncratic associations to particular music (Hernandez, Vander Wal & Spring, 2003). In general, the Music MIP is considered to be a useful tool for examining the effect of induced mood on a variety of cognitive, physiological and behavioural constructs (Clark, 1983), but it does have some serious drawbacks. Most problematic is that the Music MIP shares a confound similar to that of the Film MIP, in that a lack of consistency exists as to what music to utilize in order to induce sad moods.

A third mood induction procedure described in the literature consists of exposing participants to social interactions arranged by an experimenter. This technique has several variations. Often, participants interact with confederates who are behaving in a depressed manner (Westerman et al., 1996). Other designs ask participants to interact with a simulated "Computer Person". Participants are told they are receiving social skills training and in the depressed condition participants are informed that they have received a below average popularity rating (Martin, 1990). In general, these methods are characterized by a manipulation of social factors which allows for the induction of depressed mood without explicitly telling the participant the nature of the task. It is

assumed that these tasks elicit changes in mood based on the understanding that the behaviour of others will affect the participant's emotional state (Westerman et al., 1996). Social interaction MIPs designed to elicit sad moods have demonstrated a medium effect size of $r_m=0.437$ (Westerman et al., 1996). As with the MIPs described above, the social interaction MIPs do not offer a standardized method of mood induction, nor is there an explicit understanding of the mechanisms by which mood changes are induced.

Less common MIPs have been described by Kenealy (1986) and Westerman et al. (1996) and include hypnotic suggestion (Bower, Monteiro & Gilligan, 1978), an autobiographical recollections method using cognitive imagery to relive an experience (Brewer, Doughtie & Lubin, 1980), playing taped depressing stories to participants (Thompson, Cowan & Rosenhan, 1980), and giving participants feedback of failure (Mischel, Ebbesen & Zeiss, 1976). Interestingly, Slyker and McNally (1991) examined the possibility that mood induction manipulations are unnecessary. They simply asked participants to get into a depressed mood state, and found that some of those participants (between 50-70%) had behaviour and self report scores similar to participants in MIP conditions.

The most commonly used MIP is the Velten Mood Induction Procedure (VMIP, Velten, 1968). Participants are asked to read 60 self referential statements, some of which refer to specific somatic states, some of which are self evaluative, and some of which are a combination of the two. In the classic Velten procedure, 60 separate sets of statements are designed to induce positive (e.g., "I feel cheerful and lively"), negative (e.g., "I'm discouraged and unhappy about myself"), or neutral (e.g., "there is a large rose-growing centre in Texas") moods (Velten, 1968). This method has been modified to include

statements designed to induce hostility (Engelbreton, Sirota, Niaura, Edwards, & Brown, 1999) and anxiety (Orton, Beiman, La Pointe & Lankford, 1983). By 1990, the VMIP had been used in over 90 studies (Martin, 1990). Despite the variety of methods which have been devised to induce mood in the laboratory since Velten first postulated the idea, the VMIP still appears to be the ‘gold standard’ of mood induction tasks. Other MIPs are more often compared to the VMIP than to any other method (Clark, 1983; Westerman, 1996; Martin, 1990). The VMIP is also “by far the most widely used MIP” (Westerman, 1996, p. 559) and the frequency with which the VMIP is employed for negative mood induction is justified (Westerman, 1996). The VMIP is also ethically less problematic than some other MIPs because deception is not required in order to elicit the desired change in mood state (Martin, 1990). This allows researchers to obtain fully informed consent from participants. As such the VMIP is the most appropriate procedure to use in order to examine individual characteristics which may contribute to susceptibility to negative mood induction.

Theoretical Models of the VMIP

In order to best evaluate the results achieved by using the Velten Mood Induction Procedure (VMIP), it is important to examine the theoretical framework through which the VMIP is conceptualized. The method by which the VMIP is believed to induce depression (and the other associated moods) is conceived within a cognitive framework. According to Beck’s (1967) cognitive model, “emotional states are directly linked to the type of cognition present (e.g. Depression is postulated to result from thoughts involving unfulfilled expectations, loss and failure)” (Martin, 1990, p. 679). As such, the VMIP is based on the belief that “negative cognitions play a causal role in producing symptoms of

depression, rather than being themselves merely a symptom of depression as has often been assumed” (Martin, 1990, p. 684). This has been called the ‘vicious cycle model’ as these cognitions are both symptoms and causes of depression (Teasdale, 1983). By asking participants to focus on self referential thoughts consistent with depression, the VMIP activates the emotional states associated with these thoughts.

In the past, there has been some discussion in the research concerning whether it is the somatic or self-devaluative content of the VMIP statements that are responsible for the mood altering effects. Frost, Graf and Becker (1979) have argued that it is the somatic content of the cognitions that are solely responsible for the induction of depressed mood. Their research compared moods induced through the original positive, negative and neutral statements created by Velten, as well as mood induced through only the somatic statements (with additional self referential somatic statements produced by the authors to yield 50 somatic statements), and only self-devaluative statements (with additional self-devaluative self referential statements produced by the authors to yield 50 self-devaluative statements). Frost et al.’s discussion includes analysis of ‘tendencies’ as indicated by ‘almost’ significant differences ($p < .10$). With respect to only their significant findings, Frost et al. did find that participants in both the somatic and self-devaluative conditions reported significantly more depressed mood than participants in the positive mood condition. In support of Frost et al.’s conclusions, following induction participants in the somatic condition were significantly more depressed than those in the neutral condition, whereas participants in the self-devaluative condition were not significantly more depressed than those in the neutral condition.

In opposition to Frost et al.'s conclusions, Riskind and Rholes (1983) found that both negative somatic and self-devaluative statements were successful in inducing depressed mood relative to neutral and elated conditions, but that the self-devaluative statements had a stronger impact than the somatic statements on the participants' tendencies to make negativistic interpretations of ambiguous situations. They also found that self-devaluative statements were more effective than somatic statements in inducing heightened accessibility to negative life experiences. Their work comparing these two types of Velten statements led the authors to conclude that "negative self-devaluative statements and negative somatic statements [produce] equally equivalent effects on mood relative to a neutral or positive induction condition, as assessed by self-reports...However the self-devaluative statements (e.g. 'I am worthless') were more potent than negative somatic statements" (Riskind & Rholes, 1985).

In 1986, Nelson compared two experimental conditions (the positive VMIP and Comedy Film, a non-cognitive condition in which participants viewed a mood-inducing film) along with two control conditions in order to demonstrate that altering mood without a cognitive component was sufficient to significantly decrease the depressive mood state of depressed and non-depressed psychiatric patients, without accompanying changes in the participant's cognitions. This research indicated that there is also an emotional component through which mood can be altered.

The most recent understanding of the VMIP's mechanism of action combines all of these components, with the understanding that the Velten technique is thought to tap into all three pathways to depression; somatic, cognitive (self-devaluative), and emotional (Martin, 1990). The cognitive route includes increased accessibility to negative cognitions

and “forms the core of three influential theoretical accounts of depression” (Martin, 1990, p. 685), the somatic route involves changes in bodily state as a result of a verbal suggestion, and the emotional route may be separate from the cognitive and somatic routes, or may be “a free choice on the part of the individual to use the somatic and or cognitive routes.”(Martin, 1990, p. 685).

VMIP Mechanisms of Action

Martin (1990) describes three models which have been used to explain the VMIP’s cognitive mechanism of action: Schema theory, Semantic Network Theory, and Fragment Theory. In Schema theory, a schema is a cluster of knowledge, assumptions and beliefs accumulated from a lifetime of experiences. Schemata are used to evaluate experiences and information and to direct behaviour. The contents of a person’s schemata are related to their emotional experiences; for example a depressive schema would consist of themes of worthlessness, guilt, self-blame and rejection (Martin, 1990). “During depression the negatively toned schemata emerge from the predepressive personalities of the patients. This view would suggest that only those people who already possess negative self-schemata will be susceptible to negative mood induction procedures.” (Martin, 1990, p. 687). As such, the VMIP statements activate depressive schemata in two ways, (1) by activating the thoughts consistent with the schema and (2) by activating the emotional states associated with the schemata, (potentially through the somatic statements). Once the depressive schema is engaged it dictates an individual’s perception of their world.

In Semantic Network Theory, all of an individual’s knowledge, emotions, and experiences are represented by a series of nodes, linked together to form a network. These

links vary in strength and speed, which dictate the association between various nodes. When one node is activated, the activation spreads to other linked nodes. Depression is conceived as a result of activation of the 'depression node' (Martin, 1990; Bower, 1981), whereby activation of associated nodes results in recall of material, events and emotions previously associated with depression. Thus, the Velten induces a depressive state by activating the cognitive nodes associated with a depressed state, which in turn activates all of the associated depressive nodes with the full gamut of emotion, memories and thoughts associated with depression. This theoretical explanation also predicts that even naturally occurring activation of one negative emotion (e.g. depression) will activate other associated negative emotions (e.g. anxiety), creating a non-specific negative mood state. This corresponds to research that has found that the negative VMIP does not induce just depression, but also produces other associated negative mood states such as anxiety (Clark, 1983; Martin, 1990; Westerman et al., 1996).

The final theory presented by Martin (1990) as a potential framework for cognitive induction of depression is Fragmentation Theory. This theory conceptualizes a person's knowledge as composed of a large number of independent network fragments, as opposed to one network (as conceptualized by Semantic Network theory). Fragmentation theory accounts for the otherwise endless activation of nodes implied by the Semantic Network Theory. Because knowledge and memory is fragmented, the same information can present in a number of independent but associated nodes. A depressed mood state is more likely to be strongly associated with negative experiences and emotions than with positive experiences and emotions. Thus, a depressed mood (either induced or naturally occurring) results in the activation of the entire fragment associated with that mood state.

All of the above theories suggest that one individual characteristic that would influence the effectiveness of the VMIP would be a person's existing cognitive framework, how much the induction statements resonate with the individual's current thoughts and neural patterns. As such, a person's mood pre-induction (which according to these theories is a reflection of their cognitive state) should be an excellent predictor of the effectiveness of the VMIP task. This potential individual difference has been examined in the literature (Clark, 1983; Cairns & Norton, 1988; Blackburn, Cameron & Deary, 1990), but other aspects of a person's self schema which may influence a person's susceptibility to the Velten task have yet to be explored in detail. Blackburn et al. (1990) found that pre-induction frequency of negative thoughts was predictive of susceptibility to the depressed Velten mood induction procedure. Cognitive theory explains this finding by suggesting that a high frequency of negative thoughts may be associated with a negative bias in attention which increases the potency of self-referential negative statements, as used in the Velten task. This theory supports the reciprocal relationship between mood and cognition "whereby depressed mood may facilitate the focussing on depressed thoughts and depressed thoughts in turn increase depressed mood" (Blackburn, 1990, p. 729). Additional support for the cognitive model of mood induction was obtained by Cash, Rimm and MacKinnon (1986) who found that emotional response to mood induction was observed when the VMIP statements were congruent with the participants' belief systems (rational or irrational), but emotional response did not occur when the statements were inconsistent with the participants beliefs about themselves. With congruence (low to moderate discrepancy), the statements would resonate with the individual and activate corresponding cognitions, nodes or schemata. With incongruent

statements, there would be no activation of internal cognitive frameworks, the individual would not accept the statements as self referential, and would not accept the statement as a credible belief.

In short, the VMIP is thought to increase accessibility to negative thoughts (Velten, 1968; Martin, 1990). Thus, an individual's congruence with the mood state and cognitions associated with the mood state that a researcher attempts to induce should be one factor which influences the participant's response to the mood induction. In addition to congruence, there are other factors which may potentially affect an individual's susceptibility to the VMIP. One crucial distinction necessary to carry out this research is that between episode schemata and vulnerability schemata. Episode schemata are defined as "the maladaptive cognitive structures that become available only after the onset of a depressive episode" (Martin, 1990, p. 691). Thus, these are variables that become available and can be measured only after mood has been induced. In contrast, vulnerability schemata are defined as individual characteristics "presumed to play a causal role in depression" (Martin, 1990 p. 691). Thus, vulnerability factors would be measured *before* mood is induced, as indicators of what may cause susceptibility to a depressed mood state.

Validity of the Velten Mood Induction Procedure

The validity of the VMIP (its ability to induce the intended mood states) has been examined in a number of ways. The following section will detail a variety of evidence pertaining to VMIP validity, including literature determining whether the reports of induced mood participants are a result of demand effects, and research that has examined the content of the VMIP statements. Validity of the VMIP has also been examined with

regards to the type of measure used to assess the desired mood state, while other researchers have focused on finding differences between the neutral, elation and depressed conditions on a number of dependant variables in order to determine the extent to which the VMIP is able to influence behaviour, cognition and mood.

Demand effects.

Orne (1962, p. 779) defines the demand characteristics of an experiment as “the totality of cues that convey an experimental hypothesis” to the participants, and therefore become determinants of their behaviour. These cues include rumours about the research, information conveyed during solicitation, the conduct of the experimenter and experimental setting, and all of the explicit and implicit communications during the experiment. Depending on the method used, the VMIP can contain both explicit (in some methods the participant is asked to ‘get into’ the desired mood state) and implicit (the content of the statements indicate the desired mood state) information which allows the participant to be aware of the experimental hypothesis. It is generally assumed that demand characteristics imply that participants will want to play the role of ‘good participant’ and help validate the experimental hypothesis (Orne, 1962); as such they may exacerbate their behaviour to mimic a depressed state. Since the creation of the VMIP, experimenters have been concerned that this is the case.

In his original work developing the task, Velten (1968) found that 60 self-referential statements of either a positive (“this is great-I really do feel good- I am elated about things”), negative (“I have too many bad things in my life”), or neutral (“Utah is the Beehive State”) nature were able to induce the corresponding mood states in female college students. Significant differences between the positive and negative mood groups

were found on the Multiple Affect Adjective Check List - Today form; measures of writing speed (with positive writing more words in one minute than neutral condition participants, who in turn wrote more words than the negative mood condition participants); time needed to make decisions (with positive mood participants taking less time to make decisions than neutral and negative mood participants); and word association tasks (with negative mood participants having longer reaction times than positive and neutral mood participants). A measure of primary suggestibility (using the Harvard Group scale of Hypnotic suggestibility - Form A) was obtained prior to mood induction in order to examine the influence of this participant characteristic on the induction of mood states. Results indicated that for some tasks the more suggestible the participant the more pronounced the induced mood state, but an ANOVA examining primary suggestibility and pre-treatment mood level as covariates found that the induced mood produced significant differences among groups even after those confounds were removed. Velten also compared these experimental groups to two groups instructed to mimic positive- or negative- induced moods in order to examine the possible effect of demand characteristics on the validity of the mood induction task. Neither the positive nor negative demand characteristic groups were able to successfully mimic their respective mood states, providing evidence that the results garnered from a mood induction task are a result of a genuine shift in mood, and not of demand characteristics.

Though Velten concluded that demand characteristics were not responsible for the mood effects, Buchwald, Strack and Coyne (1981) challenged Velten's assumption on the grounds that there were not equivalent demands between the demand control and experimental conditions. Results from a replication of Velten's study with this corrected

did lead Buchwald and colleagues to conclude that “the use of the Velten induction procedure to manipulate mood experimentally or to test the relationship between cognition and mood is unwarranted” (1981, p. 478). In turn, Polivy and Doyle (1980) criticized the procedure used by Velten (1968) and Buchwald et al. (1981), suggesting that their demand characteristic groups did not provide an adequate test of whether it was demand characteristic or actual induced mood producing the effects. Polivy and Doyle compared student participants in five groups, the standard elation, neutral and depressing VMIP conditions as well as two demand conditions, where participants were asked to fake either a depressed or happy mood. They concluded that the mood shifts induced by the VMIP were, in part, a result of demand characteristics, but that the shifts in mood were not totally artificial, and that the VMIP did produce some true shifts in mood.

Westerman, Spies, Stahl and Hesse (1996) also concluded that demand effects can account for some, but not all of the effect of the VMIP. Using an experimental design similar to that of Polivy and Doyle’s (comparing standard induction conditions to participants instructed to emulate depression and elation), Alloy, Abramson and Viscusi (1981) concluded that their findings “could not be attributed to demand characteristics because nondepressed and depressed women instructed to simulate depression and elation, behaved differently than their respective mood induction groups” (p. 1129). A recent article by Finegan and Seligman (1995) critiquing both Polivy and Doyle (1980) and Buchwald et al. (1981) concluded that “the Velten technique is an effective way to induce mood” (p. 418) by demonstrating that only participants who read the VMIP statements reported mood changes. Participants given the neutral VMIP cards and

instructed to experience either a negative or positive mood state did not report a change in mood.

Martin (1990) offers further evidence suggesting that results obtained from the VMIP are not due to demand characteristics. Martin reviewed research which examined behaviour that people would most likely not emulate, such as differences in corrugator eye movements between depressed and elated Velten mood induced groups. Sirota and Schwartz (1982) found that bilateral level of corrugator muscle activity was higher for participants given the Velten depressed mood induction than participants given the Velten neutral or positive mood induction. Brown, Sirota, Niaura and Engebretson (1993) measured serum cortisol and growth hormone levels at regular intervals and found that the VMIP “induced the desired moods and that elation and sadness were associated with endocrine concomitants” (p. 458). Significantly reduced speed on a number of psychomotor tasks was also observed in depressed, but not neutral or elated VMIP conditions (Martin, 1990). In addition to these difficult to fake mood influences, Blackburn et al. (1990) concluded that depressed mood as induced by the Velten task was not a result of experimental demand effects, due to the fact that related emotions (such as deactivation, pleasantness, activation and nonchalance) all changed in the directions expected if depressed mood was experienced.

Other evidence refuting the demand characteristic explanation for VMIP induced mood is the occurrence of mood-consistent behaviour when the participants do not expect to be observed. Coleman (1975) found that participants receiving either the negative or positive mood induction could be distinguished outside of the laboratory based on general demeanour, even though the participants thought the experiment was finished.

Demand effects are said to be most likely to occur if participants are explicitly told what mood state they are to enter (as is the case with the classic VMIP). In their meta-analysis of the VMIP, Larsen and Sinnett (1991) and Westerman Spies, Stahl and Hesse (1996) found that the mean effect of the VMIP was larger when the participants were given explicit instructions concerning the mood state the experimenter was trying to induce. However, many researchers argue that this is not irrefutable proof of demand characteristics playing a part in the VMIP (Finegan & Seligman, 1995). Clark (1983) argues that by asking participants to try and feel the induced mood, the VMIP manipulation is allowing the participant to choose the means through which they want to achieve that mood state. The participant does experience the desired mood state, as a result of their decision to reach that state based on the experimenter's instruction. Westerman, et al. (1996) suggest that giving participants explicit instructions concerning the desired mood state helps cut down on interfering thoughts dealing with the purpose of the procedure, which allows the participants to devote more cognitive resources to the induction procedure and really get into the mood state.

Suggestibility.

In the original study validating the VMIP, Velten (1968) measured suggestibility using the Harvard Group Scale of Hypnotic Suggestibility to serve as a covariate in the analysis of the treatment effects. Using 100 female students, Velten found that the higher a participant's pre-induction level of suggestibility the more the positive and negative induction conditions affect them. Blackburn, Cameron and Deary (1990) found similar results using the Rod and Frame test as a measure of suggestibility. Participants with

higher suggestibility were more depressed by the negative induction procedure than participants with lower levels of suggestibility.

Effect sizes.

A meta-analysis by Westerman, Spies, Stahl and Hesse (1996) examined the size of the effect produced by a variety of mood induction procedures, including the VMIP. Results indicate that the VMIP is significantly more effective for negative than positive mood induction. For positive induction, effect sizes range from $r_m=0.49$ when measured by the Depression Adjective Check List (Lubin, 1967) to $r_m=0.3$ when measured by the Multiple Affect Adjective Check List (Zuckerman & Lubin, 1965), to $r_m=.23$ when measured by behavioural means. For negative induction, effects sizes range from $r_m=.65$ when measured by the Depression Adjective Check List to $r_m=.49$ when measured by the Multiple Affect Adjective Check List and $r_m=.26$ when measured by behavioural data. The authors conclude that, compared to other mood induction procedures, the VMIP attains high effectiveness for negative mood induction, but does not provide the most effective positive mood induction when compared to the Film/Story mood induction procedure. These results are consistent with reviews conducted by Gerrards-Hesse, Spies and Hesse (1994) as well as a meta-analysis conducted by Larsen and Sinnett (1991). In their examination, focused solely on the VMIP, Larsen and Sinnett found a large mean effect size of $d=0.76$ for the negative mood induction procedure. As such the VMIP is an excellent tool to utilize in order to investigate induced negative mood states.

Non-specific mood states.

A number of literature reviews have reported that negative mood induction procedures elicit changes in mood states other than depression (Clark, 1983; Martin,

1990; Westerman et al., 1996). Consistently, the negative VMIP has been found to have a large effect on depressed mood states, and also a medium effect on related emotions such as anxiety and hostility. In contrast, the induction of positive mood states has been found to have the same magnitude of effect on depression, hostility and anxiety dimensions (as measured by the MAACL). This may be a result of the MAACL being insensitive to positive emotional states (Westerman, et al., 1996).

According to Westerman et al. (1996), “one emotion can almost instantaneously elicit another emotion that amplifies, attenuates, inhibits or interacts with the original emotional experience” (p. 561). In other words, naturally occurring mood states do not consist of one pure mood, and thus induced mood states are expected to tap into a variety of related emotions. The existence of related mood effects also provides support for the validity of the VMIP with respect to demand characteristics. As stated by Westerman (1996) demand characteristics cannot explain why a negative mood induction fosters not only depression but also anxiety and hostility mood states that are not asked for by the examiner. The intensity, direction and type of the peripheral mood states affected by the negative VMIP varies in the literature. Clark (1983) reviewed four studies which found elevated levels of anxiety after the depression induction, as well as five studies which failed to find elevated levels of anxiety after the depression induction. Clark also reviewed three studies which found elevated levels of hostility after induction and two studies which failed to find elevated levels of hostility after negative mood induction. In all instances, if the negative induction affected peripheral emotions, the effect was smaller than those observed for depression.

The VMIP statements.

Jennings, McGinnis, Lovejoy and Stirling (2000) investigated the VMIP by examining the characteristics of the positive, negative and neutral mood induction statements. Two hundred and twenty-one undergraduate students were asked to rate level of arousal and valence (degree of pleasure) of 84 VMIP statements selected from the original Velten (Velten, 1968). Results indicated significantly different arousal ratings for the neutral, positive and negative conditions. The neutral statements were found to be significantly distinct from the positive and negative statements. Jennings et al. (2000) concluded that all three conditions of VMIP statements had high internal consistency.

Dependant variables influenced by induced mood states.

The VMIP has been used to measure the effects of mood on a variety of dependant variables. The effect of mood induced via the VMIP has been studied on pain behaviour (Willoughby et al., 2002; Zelman, Howland, Nichols, & Cleeland, 1991), eating behaviour (Klein, 1996; Cavallo & Pinto, 2001; Williams, Healy, Eade & Cowen, 2002), body image (Barber, 2001), decision making (Brecher, 2001; Armitage, Conner & Norman, 1999), memory (Knight, Maines & Robinson, 2002; Kenealy, 1997), and world view (Madigan & Bollenback, 1986). Overall the research supports the use of the VMIP as a valid method of inducing depressed mood in order to examine the effects of mood state on a variety of factors (Gerrards-Hesse et al., 1994).

Reliability.

The utility of using mood induction methods across repeated trials has been somewhat neglected in the literature (Hernandez, Vander Wal, & Spring, 2003).

Typically, MIPs compare differences in mood between groups as opposed to within, and

as such the effect of repeated MIP use has not been investigated fully. Richell and Anderson (2004) found that, although administrations of VMIP combined with Music MIP induced a depressed mood at both time one to time two, intensity of the depressed mood was higher at time one than at time two. These results prompted the authors to conclude that equivalent intensity of mood could not be induced following repeated mood induction, and as such MIPs are not optimal in repeated measures designs.

VMIP vs. Naturally occurring Mild to Moderate Depressed Mood (i.e. Subclinical Depression)

The literature strongly supports the contention that the VMIP is a useful analogue for mild to moderate naturally occurring depressed mood (Martin, 1990). A number of researchers agree that Velten depression induction produces a state which is an analogue of mild, naturally occurring retarded depression (Clark, 1983; Riskind & Rholes, 1985).

Clark's (1983) review of the VMIP and comparison of the VMIP to musical induction techniques evaluated the wide range of depression related variables which have been shown to be similarly affected by both the Velten depression induction and naturally occurring subclinical depression. Clark discussed how self reported depressed mood, psychomotor retardation (as measured by count times, writing speed, word association speed, latency to retrieve past experiences, and lever pulling speed), ability to derive pleasure from events, disturbance in appetite (for high restraint participants), preference for solitary and inactive activities, indecisiveness, levels of corrugator muscle activity, pattern of hemispheric lateralization, accessibility of negative and positive memories, and non-verbal behaviour have been found to be influenced by the Velten depression induction with patterns similar to naturally occurring depression. Because the Velten

depression induction influences all of these factors in ways analogous to naturally occurring mild to moderate depression, Clark felt that the VMIP was justifiably used as a method for examining mild to moderate depression. Though disagreeing with Clark's conclusions that the VMIP does not prove Beck's cognitive model of depression, Riskind and Rholes (1983) did concur with his conclusions that depressed states induced by the VMIP are excellent analogues for naturally occurring mild retarded depression.

The literature has also examined some of the differences between naturally occurring subclinical depressed mood and induced depressed mood. These differences must be considered when attempting to draw conclusions based on VMIP procedures related to naturally occurring depression. In his review supporting the VMIP as an analogue for mild depression, Clark (1983) summarized some dependant variables that were not found to be influenced by the Velten depression induction, but are consistently found in a naturally depressed population. Clark indicated that "depressed patients often report feeling that time passes slowly" (p. 37) but research using the VMIP found no differences in time estimation on a 7.5 minute task between the depressed, neutral, and elated conditions (Polivy & Doyle, 1981; Strickland et al., 1975). Clark (1983) argued that these studies failed to detect differences between the induction conditions because naturally depressed individuals only overestimate periods longer than 30 minutes. Dilling and Rabin (1967, as cited in Clark, 1983) found that, compared to non-depressed individuals, depressives overestimated a 30 minute period, but not a 14 minute period. Mukherji, Abramson and Martin (1982) also found that participants given the Velten depression induction did not demonstrate attributional patterns characteristic of naturally depressed individuals. Clark argued that these results do not impair the validity of the

VMIP as an analogue for naturally occurring mild depression since attributional style is conceptualized as a vulnerability to depression, and not a result of depressed mood.

Research by Kwiatkowski and Parkinson (1994) also found a difference between naturally occurring mild depression (scores of 10-16 on the BDI) and induced depressed mood on a single cognitive task. "Naturally depressed subjects show no over all deficit in intentional recall of target words relative to nondepressed subjects, whereas induced-depressed subjects exhibited poorer recall than did nondepressed subjects" (p. 231).

These results suggest that the cognitive recall of induced depression (on this one task) better mimics the pattern of naturally occurring severe depression, as opposed to more moderate states of naturally occurring depression. Because their comparison groups did not have comparable mood state scores, and because Kwiatkowski used very limited selection criteria based on BDI scores for their induction group, it is difficult to generalize these results to other cognitive tasks.

A review article by Martin (1990) provided comparable mean mood scores on the Visual Analogue Scale (maximum 100) for individuals with induced depression and clinical depression. Clinically depressed individuals were depressed psychiatric patients with BDI scores of greater than 20 who met Research Diagnostic criteria for Unipolar Major Depressive disorder. Martin (1990) reported a mean score of 53.8 for clinically depressed patients, a mean score of 70.0 for clinically depressed patients during a more depressed occasion, and a mean score of 35.0 for clinically depressed patients on a less depressed occasion. By comparison, participants experiencing the VMIP depressive induction reported mean scores ranging from 43.9 to 50.8, with a mean of 47.0 for the four studies reviewed. This comparison provides two valuable pieces of information. The

first is that the Velten mood induction procedure is capable of inducing levels of reported depressed emotion “equivalent to an intermediate clinical level” (Martin, 1990, p. 678).

As such, there is strong evidence to support the claim that the VMIP is successful in inducing temporary depressed states analogous to mild, naturally occurring depression.

The second information of note is the fact that the standard deviations were not included in the analysis. As such, it is difficult to ascertain the individual effects of the VMIP.

Some participants may achieve depression scores similar to severe depression, whereas some participants are not affected by the VMIP at all. So it may be that there is confusion in the research considering the efficacy of the VMIP because researchers consider mean depression scores, or only consider the participants who meet their induction success criteria, instead of considering the individual characteristics which make some people more susceptible to the VMIP. In other words, for some participants the Velten may produce an analogue of severe depression, for some more mild depression, and for some it may have no effect at all. A person’s individual characteristics may influence this relationship.

VMIP vs. Naturally occurring Clinical Depression (i.e. Severe Depression)

Just as Clark (1983) reviewed the similarities between Velten depressed mood and naturally occurring mild depression, Goodwin and Williams (1982) reviewed the literature to examine how results obtained using the Velten depression induction can be compared to characteristics of severely depressed patients. Participants experiencing the negative VMIP were similar to severely depressed patients in four broad categories. (1) psychomotor changes found in severely depressed patients have been mirrored in negative VMIP participants, (2) verbal communication patterns (as measured by number

of silent pauses between speech) is comparable between these two populations, (3) non-verbal deficits following a negative mood induction are equivalent to those observed in a clinical population, and (4) hospitalized depressed patients recall unpleasant memories more readily than pleasant ones. Similarly the negative VMIP increases accessibility to negative memories. Their review led Goodwin and Williams to conclude that “MIP research *is* a viable model of clinical depression” (p. 381).

There is additional research to support the possibility that the VMIP may be used as an analogue of severely depressed states. Research by Cogswell (1991) found that negative cognitive biasing of self-referential information distinguished clinically depressed adolescents from adolescents with conduct disorder with depressed mood and conduct disorder without depressed mood, suggesting that negative self-referential statements are unique to clinical depression. Since, as discussed in previous sections, the VMIP is intended to induce self-devaluative statements, theoretically the VMIP is contributing to this unique cognitive state found in clinical depression. In addition, Alloy, Abramson and Viscusi (1981) found that non-depressed students made temporarily depressed using the negative VMIP gave relatively accurate judgments of having less control over an experimental task, “of the kind normally observed in naive depressed individuals” (p. 1137). In short, the negative VMIP resulted in non-depressed students having similar cognitive distortions as naturally depressed individuals.

More recent research by Kwiatkowski and Parkinson (1994) indicates that the VMIP may be successful in inducing depressed mood states that are more severe than naturally occurring mild depression in some segments of the population. Over the course of two experiments, Kwiatkowski and Parkinson examined the effect of mood on recall of

target words and descriptor words for tasks with varying degrees of elaboration.

Experiment one compared university students with BDI scores of 0-3 (non-depressed) to students with BDI scores of 10-16 (mildly depressed). In experiment one, non-depressed students gave mean Depression Adjective Checklist (DACL) ratings of 6.78 and mildly depressed students gave significantly higher mean DACL ratings of 10.6. In experiment two, participants (all of whom scored 9 or less on the BDI) were randomly assigned to a neutral or depression VMIP. In experiment two, mean DACL ratings for students in the neutral condition were 7.66 and mean DACL ratings for students in the induced depressed condition were 16.89 for induced depression. Kwiatkowski and Parkinson did not statistically compare the DACL scores between experiment one and two, but it appears that DACL ratings were higher for negatively induced mood than for naturally occurring mild depression. The researchers provide two potential explanations which may account for this discrepancy in DACL scores. The first is that participants in experiment two were aware that the experiment involved being in a depressed state (it was part of the induction procedure) and participants in experiment one did not, suggesting an explanation consistent with demand effects. Alternatively, "it may be that mood-induction procedures cause brief intense levels of depression that are capable of reducing the available capacity enough to produce deficits in recall, whereas naturally occurring depression may be too mild to have any significant effects." (Kwiatkowski & Parkinson, p. 232). Kwiatkowski and Parkinson did find differences in the patterns of recall between naturally mildly depressed and induced depressed participants. They concluded that these were a result of differences in the performance between natural and induced depression states. An alternative explanation provided by Ellis (1985) is that the differences in performance

may be a result of the effects of mild natural depression being too weak to have any evident effects on recall, whereas induced depression, having a stronger effect on mood, may better mimic more moderate to severe depression and thus be strong enough to affect recall.

In conclusion, the research supports the assertion that the VMIP provides an analogue of naturally occurring mild to moderate depression, with some evidence suggesting it may also be an acceptable analogue for more severe forms of clinical depression. To date, most research has examined the mean effect for participants subjected to mood induction (mostly those who meet cut off mood criteria; Martin, 1990), and has not focused on the individual differences which may contribute to susceptibility to the induction task.

Problems with the Velten Mood Induction Procedure

As outlined above, the Velten Mood Induction Procedure has provided researchers with a tool able to elicit a number of mood states in a controllable, measurable way. The VMIP allows researchers to induce mood states analogous to mild and moderate depressive states which provide valuable theoretical and practical information for the study of depressed mood and potentially more severe forms of depression. Despite the VMIP being used frequently in research for the past 37 years, there are still some problems with the VMIP literature that should be addressed.

The first is the lack of a single generally adopted measure of mood and mood change. The methodological issue of having a number of techniques with which to measure if mood change has occurred following mood induction has been highlighted in a number of MIP reviews (Martin, 1990; Kenealy, 1986). A variety of measures have been

used in order to ‘check’ if the desired mood state has been achieved by participants, including self-report measures, behavioural measures, and physiological measures (Martin, 1990). In their meta-analysis, Larsen and Sinnett (1991) found that the effects of the VMIP were larger when measured by self-report than by behavioural methods. Although self report scales are thought to be more susceptible to demand effects than behavioural measures (Clark, 1983), researchers argue that this result does not necessarily indicate that the VMIP is influenced by demand characteristics. Though behavioural measures are considered to be extremely useful tools with regards to measurement of mood states, researchers have argued that self report measures are also “valid indicators of mood states, as there are especially close links between mood-related thoughts and verbally expressed-feelings” (Westerman et al., 1996, p. 563). Because of the variety of techniques used to measure mood following induction, and the differing results obtained from behavioural vs. self-report measures, an appropriate assessment tool is difficult to choose.

In addition to employing a consistent standardized measure for assessing induced mood, mood induction research must define criteria that will establish whether the mood induction has been successful. Kenealy (1986) suggests that these criteria must be operationally defined in measurable terms and be consistent across research. Unfortunately “there is no single agreed upon standard by which to judge [mood induction] success” (Martin, 1990, p. 680). In some studies, a mood induction is judged to be successful if it produces at least a 10% change from pre- to post-induction (Martin, 1990); while for other studies, a 5% change in mood from pre- to post-induction is considered to indicate ‘real’ mood change (Blackburn et al., 1990). In other studies, mood

induction is considered successful if post-induction mood states reach an agreed upon level. It becomes problematic when different studies have different success criteria, as this may explain some of the inconsistencies in the literature. Unfortunately, “most studies of mood induction report the mean mood measures only for a criterion group of subjects who changed mood in the expected direction by some predesignated amount” (Martin, 1990, p. 678), and do not include the ‘unsuccessful’ participants in their results. In other words, often studies only report results for participants for whom the mood induction is successful. This would be an acceptable practise if there were no consistent differences among the participants who are not susceptible to mood induction, but there is scant research verifying if this is the case.

This leads us to one of the most problematic aspects of VMIP research - mood induction does not work on everyone. Obviously, the success rate of the VMIP task depends largely on the criteria set by the researchers to define if the desired mood was induced. This variation explains a large amount of the inconsistency in the literature. Martin (1990) reports that the VMIP technique is effective for 75% of participants, whereas Clark (1983) reports success rates ranging from 30-50%. Buchwald, Strack, and Coyne (1981) found that 50% of the depression group and 75% of the elation group reported having ‘felt’ the induced mood. Cash, Rimm and Mackinnon (1986) suggest that participants who lack a propensity for depressive ideation can negate the effects of the VMIP by dismissing the self referential statements as ‘silly’ or sick. More often than not, researchers do not include any information about participants who do not meet induction criteria (Martin, 1990). Although some participants do not respond strongly enough to meet ‘successful mood induction’ criteria, this does not mean that the VMIP does not

affect them at all. Sirota and Schwarz (1982) found that 70% of their participants reported feelings of elation or depression after the corresponding mood induction. The remaining 30% who reported not feeling any change in mood still showed similar (though smaller) facial patterns of corrugator facial activity that were found in the mood change group. As such, an investigation of the factors which contribute to individual differences in mood induction susceptibility is warranted by the literature.

Individual Differences in Susceptibility to Depressed Mood

In the last fifteen years, a revitalization of interest in the links between personality and psychopathology has occurred (Watson & Clark, 1994). This area of investigation offers an important contribution: identification of real-world correlates of many personality constructs and the identification of potential sources of co-morbidity to better understand the etiological basis of disorders (Watson & Clark, 1994). A number of personality factors and individual differences have been identified as being associated with, and predictive of, Major Depressive disorder and depressed mood in general (Enns & Cox, 1997; Farmer & Nelson-Gray, 1990).

The literature makes a distinction between two types of individual characteristics which may contribute to vulnerability to depression. "A major theoretical issue is whether cognitive predisposition represents an enduring characteristic that is always evident, or a latent characteristic that is only evident under certain circumstances" (Teasdale & Dent, 1987, p. 113). As such, there are two categories of vulnerability characteristics. The first are identifiable only during a depressed mood state and the second are existent prior to a depressed mood state. Characteristics which meet the first criteria would not be identifiable until an individual was experiencing a depressed mood. There are a number

of theories which posit vulnerabilities ‘lurking in the wings’, waiting to be activated by stressful events, depressed mood, failure, etc. In these models, when not depressed, vulnerable individuals would appear to be just like non-vulnerable individuals. When in a depressed mood or when subjected to stressful situations, their underlying vulnerabilities would emerge, exacerbating their feelings of depression and potentially leading to a clinical level of severity (Kuiper, Olinger & Swallow, 1987; Lewinsohn, Steinmetz, Larson & Franklin, 1981). For instance, a longitudinal study by Hammen, Marks, DeMayo and Mayol (1985) indicates that depressive self-schemata did not exert an ongoing effect on everyday information processing; instead the presence of depressed mood states contributed to the activation of depressed related schemata. In a separate article, Hammen, Marks, Mayol and DeMayo (1985) reviewed a number of cognitions that are thought to be depressed mood state dependant, such as self-criticism schemata activated by personal ‘failures’. Other theorists have argued that the activation of latent characteristics may play a role in dictating if an initial mood state remains mild and transient or becomes more severe and enduring (Dent & Teasdale, 1988). Such characteristics would not be apparent before mood induction, but could be examined by comparing how the characteristic changes from pre- to post- mood induction.

In contrast, characteristics which meet the second criteria of being always evident would be identifiable as vulnerabilities by measuring individual differences *prior* to the experience of a depressed mood (Martin, 1990; Hartlage, Arduino, & Alloy, 1998; Shahar, Blatt, Zuroff, Kuperminc & Leadbeater, 2004). There are a number of individual characteristics related to depressive vulnerability which research indicates are available independent of mood state (Lewinsohn, Steinmetz, Larson & Franklin, 1981; Hammen,

Marks, Mayol & deMayo, 1985; Hartlage, Arduino & Alloy, 1998). Researchers have begun examining the individual characteristics which contribute to susceptibility to the Velten depression induction. Very few studies have examined more than one characteristic, and their results usually indicate that some, but not all, of the characteristics they examined contributed to vulnerability to the VMIP.

Research to date has recognized that the Velten MIP is not effective for every participant, but most researchers have ignored this problem, merely stating that it may confound the generalizability of their results (Clark, 1983; Kenealy, 1986; Martin 1990). Individual variability in response to the VMIP should not be considered a problem, but instead an opportunity to investigate individual differences in protective factors and vulnerability to depression. Research has shown that the VMIP is capable of effectively inducing desired mood states in some people. An addition to the literature would be an investigation of the relative contribution of the individual differences which affect vulnerability to mood induction. Results of such an investigation may allow for a better understanding of this widely used research tool, and may also allow researchers to identify vulnerability and protective factors to the induction of depressed mood states and naturally occurring depression, which could help pave the way for more effective treatment procedures.

A large number of variables have been linked to naturally occurring depression in the literature. Individual characteristics for which there is convincing evidence of a relation with naturally occurring depression will be discussed below.

Gender.

Gender differences in the prevalence and incidence of clinical depression have been well documented in the literature. A recent epidemiological review by Kuehner (2003) provides a comprehensive update on previous research (Culbertson, 1997; Sachs-Ericsson & Ciarlo, 2000), which shows that the prevalence of depressive disorder is higher in women than men. This pattern was consistent across a number of cultural settings (with ratios varying from 1.6 to 3.1) for both current and lifetime episodes. Culbertson (1997) elaborates on this difference by suggesting that the ratio difference is higher in developed countries than developing countries. The ratio of women to men experiencing Major Depression was higher than the ratio of women to men experiencing mild to moderate depression. Evidence also suggests that women may experience more somatic symptoms, co-morbid anxiety disorders and atypical depression than men. Rates of depression for men and women start to diverge around puberty and seem to reach their peak in young adulthood. Different rates of depression between men and women are still present at 60-90 years of age, though there are conflicting findings within this age group. Some researchers find the ratio declining after women experience menopause, while other researchers find that higher rates for women are maintained. Conflicting findings also surround the issue of the course of the disorder, with some researchers finding that relapse rates were similar between men and women, and others indicating that women are more likely to relapse than men. Incidence rates also show a similar trend, with 12-24 month incidence ratios of women to men ranging from 1.2 to 3.4. It is also interesting to note that there is no overall gender difference in diagnosis of clinical disorders, but that gender differences are related to specific disorders, with women being diagnosed with

depression and anxiety disorders more frequently than men (Sachs-Ericsson & Ciarlo, 2000).

Although the above epidemiological gender differences in depression have been noted for some time, the theoretical explanations for these differences are still very much debated in the literature. A number of theories have been put forth with which to explain the above, including artefacts, biological differences, and psychosocial factors such as differential exposure and vulnerability to stressors.

A number of artefactual explanations have been supported by the literature, such as a tendency for doctors to give more 'false alarm' diagnoses to women than men (Gater, Tansella, Korten, Tiemens, Mavreas, & Olatawura, 1998 as cited in Kuehner, 2003), gender differences in help seeking behaviour (Kuehner, 2003; Sachs-Ericsson & Ciarlo, 2000) and gender differences in the phenomenology of depression (with men experiencing more 'irritable' and antisocial features (Kendler & Gardner, 2001 as cited in Kuehner, 2003). In addition, some artefactual hypotheses which have not been supported by empirical evidence include suggestions that gender influences ability to recall past affective states (Kendler, Gardner & Prescott, 2001 as cited in Kuehner, 2003).

Biological explanations have incorporated findings which indicate that men and women share most, but not all genetic influences for Major Depression (Sullivan, Neale & Kendler, 2000 as cited in Kuehner, 2003). Studies have identified a relationship between levels of sex hormone, social factors and negative affect in women (Hankin & Abramson, 2001 as cited in Kuehner, 2003), and gender differences in hormonal responses to stress (Young, 1995 as cited in Kuehner, 2003).

Psychosocial explanations have been supported by findings indicating that gender differences in depression are less evident in societies in which the traditional female role is valued similarly to that of the traditional male role (Piccinelli & Wilkinson, 2000 as cited in Kuehner, 2003), that gender differences in early socialization contribute to different coping styles (Sachs-Ericsson & Ciarlo, 2000), as well as examinations of the poorer social status of women fitting into a learned helplessness model (Nolen-Hoeksema & Girgus, 1994 as cited in Sachs-Ericsson & Ciarlo, 2000). It is interesting to note that age may play a role in mediating these factors. As noted by Kuehner (2003), different risk factors may have different effects on women's experience of depression across the lifespan.

A correlate of the literature theorizing explanations for gender differences in depression diagnosis is the identification of depression vulnerability factors in men and women. Galambos, Leadbeater and Barker (2004) used a longitudinal approach (4 years) to determine vulnerability factors which may differ between adolescent women and men. The researchers found no evidence that any of the risk factors they examined (including amount of social supports, body mass index, smoking frequency) were related differentially to depressive symptoms for boys and girls. The authors concluded that "further research on the mechanisms that support the notable sex differences in depression in boys and girls is clearly needed" (p. 23). Other research by Goodwin and Gotlib (2004) found that gender moderated the relationship between neuroticism and depression. A regression analysis of telephone survey data obtained from non-institutionalized Americans indicated that neuroticism significantly contributed to the relationship between gender and depression, suggesting that a higher level of neuroticism

among women may help to explain the increased prevalence of depression among women.

A relational explanation for understanding depression in women has been proposed by Jack (1991). Jack has conceptualized women's experience of depression as relating to 'silencing of their self'. This theoretical conceptualization of depression in women assumes that "women's vulnerability to depression lies in their development of a self-identity which is grounded in interpersonal experiences" (Stoppard, 2000, p. 67).

According to Jack, self silencing is a result of conditions characterized by "specific forms of unequal, negative intimate relationships as well as larger social structures that demean an individual's sense of self-worth" (Jack, 1996, p. 15, as cited in Stoppard, 2000).

"Silencing behaviours include putting the needs of others first, censoring and repressing genuine emotion, and judging themselves from external standards. Self-denial and silencing results in the eventual loss of self, and it is this loss that is hypothesized to create vulnerability to feelings or symptoms of depression in women." (Cramer & Thoms, 2003, p.526). Jack has operationalized this theory with the Silencing the Self Scale (STSS, Jack & Dill, 1992). Empirical validation of this scale was done on three groups of women; undergraduates, women in battered women's shelters and women in a cocaine and pregnancy health study. Internal consistencies ranged from .86 (for the undergraduate group) to .94 (for the shelter sample) (Jack & Dill, 1992). Test-retest validity was in a similar range. Results supporting construct validity include significant correlations between STSS score and mild and moderate depression, with participants with higher STSS scores reporting higher scores on depressive symptom measures (Jack & Dill, 1992; Stoppard, 2000). In addition, women with a history of sexual/physical abuse have

higher STSS scores than women without a history of abuse (Stoppard, 2000). In sum, the STSS scale provides a unique and empirically validated measure that allows an examination of vulnerabilities to depression in women.

Some research has found that women are more susceptible to the effects of the Velten mood induction than men (Gouaux & Gouaux, 1971; Strickland et al., 1975; Lewis, Dember, Schefft & Radenhausen, 1995), whereas other research has found that there were no differences in the responses of men and women to the VMIP (Blackburn, Cameron, & Deary, 1990; Buchwald et al., 1981; Cairns & Norton, 1988; Lewis & Harder, 1988). In their meta-analysis, Westerman et al. (1996) found that the gender of participants did not contribute to the effect size of VMIP. As such, there is some inconsistency in the literature regarding gender differences in response to the VMIP.

In short, gender, for whatever biological, social, or other reason, is thought to play a role in the experience of depression, though the exact nature of that role is still unknown. In addition, the literature provides conflicting evidence as to whether gender plays a role in participants' responses to depressed mood induction tasks. What the literature does suggest is that there may be a gender difference in the factors which contribute to vulnerability to depression (Goodwin & Gotlib, 2003). Due to the gender difference in diagnosis of depression, an understanding of the individual differences which contribute to naturally occurring depression in women and men seems warranted. As such, an examination of a variety of personality characteristics which contribute to susceptibility to induced depressed mood in women, as compared to men, will provide a valuable and timely contribution to the literature.

Locus of control.

Locus of control has been conceptualized as a continuum, ranging from internal to external focus. Individuals with an internal locus of control believe that they are capable of controlling events, whereas individuals with an external locus of control believe that events are outside of their control (Lamanna, 2001; Clarke, 2004). Benassi, Sweeney, and Dufour's (1988) meta-analysis demonstrates a consistent, moderately strong relationship between an external locus of control orientation and depression. Recent evidence provides further support for this relationship. Lamanna (2001) found that an external locus of control was a significant predictor of depression. Jaswal and Dewan (1997) found a moderate correlation between external locus of control and depression in college students and concluded that "there is a circular link between locus of control and depression. Depression can be both an effect and a cause of external locus of control" (p. 27). Longitudinal analysis over a six to nine month period indicates that a higher internal locus of control predicted transient versus persistent depression in a sample of African American women (Mentes, 2004). In their meta-analysis of the relationship between depression and locus of control Benassi et al.(1988) drew attention to a paradox in the depression literature, that depressed individuals seem to perceive events beyond their control (Seligman's helplessness theory), while at the same time blaming themselves for failures (Beck's negative schemata theory). Clarke (2004) attempted to resolve this paradox by investigating the role of neuroticism in the relationship between locus of control and depression. Using a path analysis, Clarke found evidence that neuroticism mediates the relationship between external locus of control and depression.

Natale's (1978) findings also support a rationale for examining the effect of locus of control on vulnerability to induced depressed mood. Natale used the Velten Mood Induction Procedure to induce randomly assigned depressed, neutral, or positive mood states in 45 female college students. Natale measured locus of control both pre- and post-induction, and analyzed the data to examine change in locus of control to compare between the negative, positive and neutral condition. Natale did not analyze how pre-induction locus of control contributed to Velten vulnerability, but he did find that depressed participants experienced increased external locus of control, while the elated groups experienced increased internal locus of control as compared to their pre-induction levels.

Despite the theoretical and correlational relationships between depression and locus of control, locus of control has not been found to be a predictor of vulnerability to induced depressed mood. Lewis and Harder (1988) examined locus of control by dividing the responses of 51 male and 39 female university students into internal and external locus of control using a median split. They found that pre-induction locus of control (as measured by Rotter's Internality-Externality Scale, 1966) did not contribute to their participants' mood responses to randomly assigned positive, neutral, or negative VMIP conditions. Cairns and Norton (1988) found similar results when they examined pre-induction mood (as examined by the profile of mood states) and pre-induction locus of control (as measured by Rotter's Internality-Externality Scale, 1966). Cairns and Norton generated their own self-referent statements to induce neutral, anxious, hostile and depressed mood (60 statements for each mood). Locus of control was hypothesized to be predictive of mood induction susceptibility because it was theorized that participants with

an internal locus of control may be more resistant to external manipulation, whereas participants with a more external locus of control would “expect control and therefore be less resistant” (p. 416). After measuring locus of control and pre-induced mood, 40 participants (20 women) were randomly assigned to experience either the anxious, hostile or depressed mood induction and asked to rate their mood on the MAACL. Results indicated that internal/external locus of control was not significantly correlated with susceptibility to any of the mood states tested. A post-hoc regression analysis conducted on theoretically derived self referential statements designed to induce depressed mood by Rexford and Wierzbicki (2001) also found that locus of control (as measured by Rotter’s Internality-Externality Scale, 1966) did not significantly predict response to negative mood induction for 84 college students.

The contrast in the relationship between locus of control and naturally occurring depression vs. locus of control and induced depressed mood suggests that further investigation of the relationship between locus of control and induced depressed mood is warranted. Two potential explanations exist for this discrepancy. The first is the small sample size employed by the studies examining the relationship between locus of control and susceptibility to induced depressed mood. The second may be the tool used to measure locus of control, namely Rotter’s Internality-Externality Scale (RLOC), which was used in all three studies examining induced mood susceptibility. Recently this scale has been criticized for being moderately related to measures of the theoretically separate constructs self efficacy and interpersonal power (Leone & Burns, 2000). In addition, the RLOC has been criticized for being “a poor measurement instrument” with minimal internal consistency, a poor rationale for its forced-choice format, a false assumption of

unidimensionality, and “indistinguishable facets which cannot be inferred with sufficient reliability to be practically useful” (Marsh & Richards, 1984, p. 1). The use of a different measure of locus of control may demonstrate a relationship between external locus of control and susceptibility to induced depressed mood.

Emotional intelligence.

Emotional Intelligence (EQ) has been classified into two separate dimensions. The first is EQ ability, which is defined as “the ability to perceive accurately, appraise and express emotion; the ability to access and/or generate feelings when they facilitate thought; the ability to understand emotion and emotional knowledge; and the ability to regulate emotions to promote emotional and intellectual growth” (Warwick & Nettelbeck, 2004, p. 1092). EQ ability is measured by performance. The second is EQ trait, which can be described as emotional self efficacy (Warwick & Nettelbeck, 2004). EQ trait is measured by self report. Preliminary evidence indicates that measures of EQ ability largely overlap with measures of the EQ trait and vice versa (Petrides & Furnham, 2003).

EQ has been identified as a protective factor against depression. In other words, individuals high in EQ are less likely to suffer from depression, whereas individuals low in EQ are more likely to suffer from depression (Lamanna, 2001). Other research suggests that high EQ may help individuals manage life stress, and thus prevent feelings of depression and suicidality (Ciarrochi et al., 2002).

Recent research also indicates that EQ may provide a protective factor to the experience of induced depressed mood. Schutte et al. (2002) investigated how emotional intelligence (measured using the Emotional Intelligence Scale, Schutte, 1998) related to

induced mood susceptibility by examining the effect of Emotional Intelligence on state mood scores and self esteem scores after negative and positive Velten Mood Induction. Forty-seven participants were given self report questionnaires to measure state self esteem (measured by adjusted Rosenberg self esteem scale), state mood (measured by PANAS) and EQ. Participants were then given either the negative or positive VMIP then their self esteem and mood was reassessed. Results indicated higher EQ scores provided a protective barrier (participants were less likely to report a decrease in positive mood as measured by the PANAS) after experiencing the negative Velten mood induction task. Higher EQ was also associated with less decrease in self-esteem after the negative VMIP. Participants with higher EQ also reported a greater increase in positive mood after the positive VMIP.

In contrast, Petrides and Furnham (2003) found that participants with high trait EQ (in the top 15 percentile, 17 participants) were more sensitive to changes in some mood states induced using MIPs than participants with low trait EQ (in the bottom 15th percentile, 17 participants). Petrides and Furnham used the BarOn EQ-I (Bar-On, 1997) scale to measure EQ and added their own subset of questions to ensure “covering some of the important trait EI facets that the EQ-I does not” (p. 4). Using a film MIP, Petrides and Furnham induced depressed mood and then positive mood in all participants and measured changes in nine different mood states. Results showed that the high EQ groups showed statistically more mood change than the low EQ group on measures of tension, anger, confusion, and vigour. With respect to a depressed mood though, the high EQ and low EQ group were not significantly different in their responding.

One potential explanation for the conflicting findings with respect to EQ and mood induction is that Petrides and Furnham statistically partialled out the effects of the ‘Big Five’ personality variables (Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness as measured by the Revised NEO personality inventory (Costa & McCrae, 1992)) before investigating the effect of EQ on induced mood susceptibility, whereas Schutte et al. did not. Further investigation of the relationship between EQ and induced mood seems warranted by the differing results in the literature. These conflicting results highlight differences in the conceptualization of EQ. Schutte et al.’s results support EQ as a protective factor, a characteristic which allows for better internal management of emotions. Petrides and Furnham’s null findings indicate that high EQ does not allow individuals to protect themselves against negative mood induction. A regression analysis including characteristics similar to the ones partialled out by Petrides and Furnham will allow for resolution of this controversy by indicating the relative contribution of EQ to induced depressed mood vulnerability.

Neuroticism.

Neuroticism has been criticized as such a general description of distress and dysfunction that alone it is not useful (Claridge & Davis, 2001). Hierarchical regression analyses by Cox et al. (2004) demonstrated that neuroticism did not predict Major Depression in a sample of 5877 people when socioeconomic status, history of anxiety disorders, substance abuse and current emotional distress were controlled for. These results support the conclusion that neuroticism alone is a general correlate of distress as opposed to a specific predictor of Major Depression. Examinations of neuroticism support this criticism by demonstrating that neurotic traits are associated with any clinical

disorder involving high levels of subjective distress (Watson, Gamez & Simms, 2005). That said, neuroticism is a potentially useful construct, especially when examined along side other personality variables such as locus of control (Claridge & Davis, 2001; Schmitz, Kugler & Rollnik, 2003). A number of studies have demonstrated that neuroticism is strongly associated with naturally occurring clinical depression (Watson, Gamez & Simms, 2005; Schmitz, Kugler & Rollnik, 2003) especially for women (Kendler, Kuhn & Prescott, 2004; Goodwin & Gotlib, 2003).

In addition to being associated with naturally occurring depression, neurotic traits contribute to vulnerability to induced depressed mood states. Hill (1985) examined how individual differences in neuroticism contributed to the differing effect of the Velten task on participants. With 80 female participants (university students not taking psychology with no history of psychiatric illness), Hill (1985) examined how pre-existing neurotic and extravert/introvert traits influenced susceptibility to depressed and neutral mood induction using the Velten Mood Induction procedure. Using writing speed measured pre- and post-induction to indicate level of depressive mood, Hill found that the Velten was successful in significantly lowering writing speed in the depressed mood condition, as compared to the neutral mood condition. ANOVA analysis indicated that neurotic participants (extraverted or introverted) were more vulnerable to induced depressed mood than were more stable participants. This finding, that participants with neurotic personality traits are more vulnerable to experience induced depressed mood, is consistent with other MIP literature (Blackburn, Cameron & Deary, 1990; Schreindorfer, 2002). Hill offers a theoretical explanation for his findings: the VMIP is believed to activate cognitions by increasing the participants' self-awareness. Such activation of cognitions

would include the activation of the associated mood states. Since neurotic individuals are believed to be predisposed “by personality to feel emotions more readily and strongly” (Hill, 1985, p. 525), they would be more affected by the emotions instigated by the Velten procedure.

Pessimism / negative attributional style.

Pessimism has been defined simply as “an expectation that bad things will happen” (Chang, 2001, p. 5). Research has shown that people with clinical depression have more pessimism about the future than people without clinical depression (Parker, Wilhelm & Asghari, 1998; Hartlage, Arduino & Alloy, 1998). Conversely, an optimistic cognitive style is theorized to provide a protective barrier to depression (Abramson, et al., 2000). A five year longitudinal study (Temple-Wisconsin Cognitive Vulnerability to Depression Project) examined participants in the upper (pessimists) and lower (optimists) quartiles of the Cognitive style questionnaire or the dysfunctional attitude scale. Results from this survey indicated that optimists had a significantly lower incidence of Major Depression than pessimists, whereas optimists did not differ from pessimists in their rates of other Axis 1 disorders (Abramson et al., 2000).

Findings also indicate that depressed mood states result in increases in pessimism. Powers (1997) demonstrated that negative mood induction increases pessimism scores significantly more than neutral mood induction. Lewis, Dember, Schefft and Radenhausen (1995) found similar results by examining whether optimism and pessimism scores were influenced by a positive or negative mood state (as induced by a Velten MIP). Lewis et al. used the Optimism/ Pessimism (O/P) Scale developed by Dember et al. (1989) in order to assess level of pessimism pre- and post- mood induction. Their

investigation focused on the change in optimism and pessimism scores; in other words, pessimism was the dependant variable and mood state was the independent variable. Their results indicated that women responded with less pessimism following the positively valenced inductions and more pessimism following the negatively valenced inductions. This contradicted previous findings by Terezis (1990) who found that pessimism scores were stable pre- and post- negative mood induction. Lewis et al.'s results suggest an interesting relationship between pessimism and depression, in that a depressed mood state increases pessimism. Unfortunately, despite measuring mood state post mood induction, Lewis et al. did not analyze whether individual differences in pessimism contributed to change in mood state following the VMIP. By neglecting this analysis, Lewis et al. (1995) were unable to determine if the opposite causal relationship exists between pessimism and depression, namely that pessimism increases vulnerability to experiencing depressed mood states.

As such, further investigation is required to determine if the reciprocal relationship is true, if pessimism results in a vulnerability to induced (and therefore potentially naturally occurring) depressed mood. Pessimism can also be operationalized as a negative attributional style (Chang, 2002). Cash, Rimm and MacKinnon (1986) suggest that research should examine susceptibility to mood induction as a function of other predisposing cognitive variables such as Seligman's negative attributional style, which has been characterized as a *pessimistic* style of interpreting events. Lewinsohn, Steinmetz, Larson and Franklin (1981) also highlight Seligman's negative attributional style as a cognitive characteristic related to depressive vulnerability that is available antecedent to depressive mood states. Using a two stage longitudinal design (three

months) encompassing 68 university students, Kapçi and Cramer (2000) examined how negative attributional style (as measured by the Attributional Style Questionnaire) was able to predict depressive symptoms, in an attempt to examine a hopelessness model. Kapçi and Cramer found that participants who experienced a large (>4) number of negative life events AND who attributed those negative life events to stable, global factors (i.e. had a negative attributional style) had a greater chance of having subsequent depressive symptomatology (as measured by the BDI). Other evidence for a causal link between negative attributional style (i.e. pessimism) and clinical depression has also been found by Abramson and colleagues (1998). Examining whether pessimism scores (i.e. negative attributional style) predict vulnerability to depressed mood states may provide further information regarding the causal relationship between these two constructs.

Self critical perfectionism.

Perfectionism has been shown to be positively associated with depressed mood and in longitudinal studies perfectionism has been linked to both depression and hopelessness over time (Wei, Mallinckrodt, Russell, & Abraham, 2004; Enns & Cox, 1997). However, in general research involving the link between perfectionism and depression is lacking due to new conceptualizations of perfectionism in the field (Enns & Cox, 1997). In a recent review Enns and Cox (1997) identified the relationship between perfectionism and depression as a promising area for further research. Recently, perfectionism has been re-defined from a unitary construct to a new conceptualization as two distinct types of perfectionism: maladaptive and adaptive (see Bieling, Israel & Antony, 2004 for a review of the evidence to support this separation). Adaptive forms of perfectionism foster excellence and striving to meet important goals whereas maladaptive

perfectionism is conceptualized as “related to negative reinforcement and self defeating behavior. For instance, being concerned about how others evaluate the self, self-doubt, and worry about making mistakes” would be considered maladaptive perfectionism (Bieling, Israel & Antony, 2004, p.1375). The Dysfunctional Attitudes Scale (Weissman & Beck, 1978) has been used successfully to assess cognitive aspects of perfectionism (Powers, Zuroff & Topciu, 2004)

Maladaptive perfectionism has been further refined to include the concept of self-critical perfectionism (see Dunkley & Blankstein, 2000 for a more detailed explanation of this construct). Self-critical perfectionism is described as maladaptive perfectionism with “punitive and self-denigrating component[s] as well as ... hypersensitivity to perceived excessive external standards and criticism” (Powers, Zuroff & Topciu, 2004, p. 62). Using structural equation modelling on data from college students, Powers, Zuroff and Topciu (2004) found that covert self-critical perfectionism was an important predictor of depressive symptoms (as measured by the BDI). Dunkley, Zuroff and Blankstein (2003) also demonstrated that self-critical perfectionism was associated with high negative affect due to effects on stress and coping. A recent hierarchical regression analysis of national survey data by Cox et al. (2004) indicates that self-criticalness is an excellent predictor specifically of Major Depression. Because self critical perfectionism is a relatively new construct (Dunkley & Blankstein, 2000; Dunkley, Zuroff & Blankstein, 2003; Powers, Zuroff & Topciu, 2004), research has yet to examine how self critical perfectionism influences susceptibility to induced negative mood, though the empirical evidence reviewed above suggests a connection is likely.

Rumination.

Rumination has been defined as “the tendency to engage in repetitive negative thinking” (Papageorgiou & Siegle, 2003, p. 243). Papageorgiou and Siegle (2003) highlight rumination as a current ‘hot topic’ in depression literature, with publications with ‘rumination’ in the title increasing linearly over a 17 year period and interest in the topic spanning disciplines to include research by social psychologists, health psychologists as well as clinical research. It is hypothesized that a negative cognitive style (negative attributions, dysfunctional attitudes) coupled with rumination is likely to result in clinical depression (Alloy et al., 2000; Robinson & Alloy, 2003). This theory has received empirical support, with results demonstrating a significant interaction between negative cognitive style and a tendency to ruminate on negative inferences following stressful life events in their ability to predict retrospective lifetime rates of major depressive episodes (Alloy et al., 2000). Using a similar design, Robinson and Alloy (2003) also found that stress-reactive rumination combined with negative cognitive style (as measured by the Cognitive Style Questionnaire and Dysfunctional Attitudes Scale) predicted depression symptomology, further demonstrating that rumination is a valid “etiological factor in the onset of depression” (p.289). In addition, Just and Alloy (1997) found that “nondepressed (ND) participants who reported that they ruminate in response to their depressive symptoms were more likely to experience a DE [depressed episode] over 18 months than were participants who reported that they distract themselves from their symptoms” (p. 221). A four month longitudinal study by Kuehner and Webber (1999) found a similar pattern of results for patients with Major Depression, resulting in their conclusion that “rumination is likely to have a deteriorating impact on the course of

clinical episodes of depression in unipolar depressed patients”(p. 1323). Despite the bulk of evidence linking rumination to naturally occurring depressive symptoms and clinical depression, to date rumination has not been examined as a predictive factor in susceptibility to induced depressed mood. Such an examination could provide further information concerning the causal nature of the relationship between rumination and depression.

A Model Study

Blackburn, Cameron and Deary (1990) examined the effect of pre-induction neuroticism, extraversion, negative mood, frequency of negative thoughts, suggestibility and gender on vulnerability to induced depressed mood, using the VMIP. Using 40 university students (20 women, 20 men) Blackburn et al. attempted to determine why the VMIP is only successful for approximately 50 percent of participants (Clark, 1983; Polivy & Doyle, 1980) by examining the individual differences that may contribute to vulnerability to the VMIP (and potentially vulnerability to naturally occurring depression as well). Using a visual analogue scale to measure severity of induced depressed mood, results indicated that neuroticism (as measured by the EPI), pre-induction level of depression (as measured by BDI), frequency of pre-induction negative thoughts (as measured by Automatic Thoughts Questionnaire), suggestibility (as measured by the Rod and Frame Test), experience of recent negative events (as measured by self-report), and degree of belief in the Velten statements (as measured by a Visual analogue scale with poles “not at all” and “very much”) predicted responses to the negative Velten mood induction. Thus, Blackburn and his colleagues were able to verify personality factors that contribute to the variability in susceptibility to the Velten procedure. Though this was an

excellent beginning, Blackburn et al.'s work was preliminary, and as such further research is required in order to further understanding of these individual differences. Blackburn's article calls for "future research with larger groups of subjects, using multiple regression analysis" (p. 729). In addition, the individual traits Blackburn looked at were dictated by the cognitive and social theories of 1990. As such, more recent understanding of the variables which contribute to naturally occurring depression provides theoretical and empirical reasons to examine a number of traits neglected by Blackburn et al.

Summary and Conclusions

In summary, it is clear that some characteristics which are related to naturally occurring depression are also predictors of vulnerability to induced depressed mood (such as neuroticism). Yet the literature also indicates that this is not always the case; some characteristics which are thought to be related to clinical depression have been found to have no influence on induced depressed mood vulnerability (such as locus of control). Also of interest are characteristics which demonstrate inconsistent ability to predict susceptibility to induced depressed mood (such as gender and emotional intelligence). Other characteristics which have been strongly linked to naturally occurring depression have yet to be examined with respect to predicting vulnerability to induced depressed mood (such as self-critical perfectionism and rumination). In addition to replication of findings and clarification of inconsistent results, future research using multiple regression analysis will be able to yield what, to date, has been largely ignored in the VMIP literature: the relative contribution of each variable to depression vulnerability. As described above, most studies examining individual differences which contribute to

VMIP susceptibility have looked at one or two characteristics in isolation. Recent investigations have focused on determining how more than one individual difference interacts to create vulnerability to naturally occurring depression (Clarke, 2004; Wei, Mallinckrodt, Russell & Abraham, 2004). Additional research is needed to examine the relative contributions of those individual characteristics which relate to naturally occurring depression to VMIP vulnerability using a multiple regression analysis.

Methodological Issues in the Literature

There are a number of key methodological issues apparent in the mood induction literature. One methodological issue is whether to use change scores or only post-induction mood scores to determine mood induction success. A second problem is the common practise of measuring mood states immediately prior to mood induction. Finally, an examination of the 'scar' hypothesis provides a theoretical reason to utilize non-depressed participants in an examination of depressive vulnerabilities.

Change scores as a measure of mood induction success.

In his original paper championing the VMIP as a useful tool able to successfully induce depressed mood states which are valuable analogues to naturally occurring depression, Clark (1983) strongly recommended that mood change scores (pre- to post-induction), rather than mood scores following mood induction, be used to assess the efficacy of mood induction techniques. Though some researchers have argued against using change scores in a regression analysis (Cohen & Cohen, 1975), more recent work has argued that using change scores in a regression analysis is not only possible, but is the preferred type of dependent variable to use in a regression analysis (Allison, 1990).

Despite these recommendations, many procedures have assessed the effects of mood induction based solely on participants' mood scores following mood induction. This procedure makes some theoretical sense when comparing mood changes between conditions (i.e. employing the use of a neutral vs. control conditions) as was done by Mukherji, Abramson and Martin (1982) as well as Schare and Lisman (1984). However other experimenters have not used a control group, and have still measured mood induction success using only post-induction mood scores. This issue becomes most pertinent when attempting to measure individual differences in response to the VMIP. Additional arguments supporting the need to examine change in mood, as opposed to mood following induction, can be made for the examination of the effects of pre-induction mood states on post-induction mood.

Pre-induction mood.

The literature provides conflicting information with respect to pre-induction mood as a predictor of post-induction mood. Velten (1968) examined pre-induction mood using measures of Decision Time and Perceptual Ambiguity in order to covary the effects of pre-induction mood from his experimental findings. Results indicated that "pre-treatment mood level was essentially unrelated to any treatment subjects' criterial performance" (p. 477). Cairns and Norton (1988) also examined whether pre-induction mood (as examined by the Profile of Mood States) influenced susceptibility to induced anxious, hostile or depressed mood. Pre-induction mood was hypothesized to predict post-induction mood. Forty participants (20 women) were randomly assigned to experience either the anxious, hostile or depressed mood induction and asked to rate their mood on the Multiple Affect Adjective Checklist. Results indicated that pre-induction mood was correlated with

hostility score after an angry mood induction, but was not significantly correlated with mood after the depression or anxiety induction. Conflicting results were found by Blackburn, Cameron and Deary (1990) using 40 undergraduate students. Blackburn and colleagues found that pre-induction level of depression (measured by BDI) and frequency of negative thoughts predicted susceptibility to induced negative mood. The more depressed participants were before the induction, the more vulnerable they were to the negative mood induction. Similarly, the more negative thoughts participants identified with at pre-induction, the more severe their response to the VMIP. Goff (1999) also found that pre-induction mood predicted mood following a negative film MIP.

In summary, there is inconsistency in the literature regarding whether pre-induction mood predicts post-induction depression scores. Further examination of this relationship would be useful to both clarify discrepancies in the literature and to covary out the effects of pre-induction mood in order to obtain a better picture of other factors related to induced mood vulnerability (Clark, 1983).

Measuring mood states immediately prior to mood induction.

Two studies have provided pertinent information dictating a re-examination of the methodology typically used in mood induction experiments. A number of studies measure pre-induction mood immediately before presenting the mood induction task, often using the MAACL to measure mood (Lewis & Harder, 1988; Cash, Rimm & MacKinnon, 1986, Lewis et al., 1995). This is a common procedure in the literature. Unfortunately its effects on subsequent mood induction and mood measurement have not been thoroughly explored.

Research by Nagata and Trierweiler (1988) demonstrated that giving participants the Multiple Affective Adjective Checklist (MAACL) immediately prior to the negative VMIP resulted in a significant increase in the MAACL anxiety scale post-induction, as compared to participants who were not tested prior to induction. Nagata and Trierweiler also report evidence suggesting that mood testing (using the MAACL) immediately prior to mood induction can influence depression scores. Informal analysis of previous VMIP research using the MAACL indicated that “studies using mood pre-tests found smaller mean post-induction depression score differences between the Velten-D [negative VMIP] and Velten-N [neutral VMIP] groups than did studies not using these pre-tests. The pre-test studies reported no more than a 6-point difference, whereas the no pre-test studies reported differences of 10 or more points (Nagata & Trierweiler, 1988, pp. 132-133). These results led Nagata and Trierweiler to recommend that “researchers include non pre-tested control groups in future studies to assess the impact pre-testing might have upon their results” (p. 133). Unfortunately this solution is not applicable to an investigation of individual differences.

Similar effects have been found for another common measurement of depression which is also often presented immediately following mood induction, the Beck Depression Inventory (BDI). Mark, Sinclair, and Wellens (1991) found that non-depressed participants given a BDI are more likely to report a more positive mood than non-depressed participants not given a BDI prior to a self report mood scale. In contrast, individuals with a history of clinical depression given the BDI first are more likely to report a more negative mood than similar individuals not given a BDI prior to completing a self report mood scale. In other words, asking someone to rate their depressive

symptoms immediately prior to reporting on their mood influences self report mood ratings.

These results must be considered when attempting to measure characteristics prior to measuring mood state. Mood state must be measured prior to mood induction in order to obtain a change in mood score (which the literature indicates is necessary), but assessing mood immediately prior to mood induction influences mood state. If asking individuals about their mood state and depressive symptoms changes their mood state (artificially or otherwise), how can a paradigm be designed to obtain a clear picture of the effects of negative mood induction without ignoring contributing factors? The strategy that seems warranted is employing a time delay between pre-induction mood measure and Velten mood induction procedure. In the present study, participants will be given a ten minute rest period between measurement of mood state and personality characteristics and induction of depressed mood in order to counteract the effects of assessing mood and personality on the induced mood state.

The 'scar' hypothesis.

An examination of the 'scar' hypothesis provides a theoretical reason to utilize non-depressed participants in an examination of depressive vulnerabilities. The scar model of depression hypothesizes that depression alters a person's personality (Shahar et al., 2004). As such, examining the personality characteristics of individuals who have previously been depressed (or who are currently depressed) may not provide an adequate picture of the causal relation between personality characteristics and depressive vulnerability. The 'scar' model dictates that an examination of susceptibility to depression

must be undertaken using participants whose personality has not been potentially altered by an episode of Major Depression

The Present Investigation

The present investigation aims to use the VMIP to investigate the impact of various individual characteristics already shown to be related to naturally occurring depression in order to assess their relative contribution to susceptibility to induced depressed mood. This will contribute to our understanding of vulnerability to depressed mood and to our understanding of vulnerability to naturally occurring subclinical depression.

Hypotheses

H1. Women will be more susceptible to the negative Velten Mood Induction than men.

Rationale: studies which found that women were more susceptible to the VMIP than men (Gouaux & Gouaux, 1971; Strickland et al.1975; Lewis, Dember, Schefft & Radenhausen, 1995), as well as rates of naturally occurring depression which indicate that women are twice as likely to be diagnosed with Major Depression than men (Kuehner, 2003; Culbertson, 1997; Sachs-Ericsson & Ciarlo, 2000).

H2: There will be a direct relation between external locus of control and susceptibility to the negative VMIP.

Rationale: Evidence linking naturally occurring depressed states with external locus of control (Lamanna, 2001; Benassi, Sweeney, & Dufour, 1988). Surprisingly, (given the connection between naturally occurring depression and locus of control) previous research examining the relationship between locus of control and susceptibility to mood induction found no relationship between these two constructs

(Cairns & Norton, 1988; Lewis & Harder, 1988; Rexford & Wierzbicki, 2001), but results from this research are questionable, give a very small sample size (cell size of only 10 participants). Cairns and Norton's use of their own mood induction statements as opposed to the standardized Velten statements, as well as the locus of control measurement tool raises additional questions. The current study will employ a larger sample size, will use a validated (the VMIP) mood induction technique, and will employ a potentially more valid measure of locus of control.

H3: There will be an inverse relation between emotional intelligence and susceptibility to the negative VMIP.

Rationale: High emotional intelligence has been identified to be a protective factor to the experience of naturally occurring depression (Lamanna, 2001; Ciarrochi et al., 2002). Correspondingly Schutte et al. (2002) found that participants with high EQ were less susceptible to the negative VMIP. Unfortunately their results failed to take into account the contribution of other personality variables (Petrides & Furnham, 2003). A regression analysis including other variables will indicate the specific contribution of EQ suggested by the literature.

H4: There will be a direct relation between neuroticism and susceptibility to the negative VMIP.

Rationale: Research showing the connection between neuroticism and clinical depression (Watson, Gamez & Simms, 2005; Schmitz, Kugler & Rollnik, 2003; Kendler, Kuhn & Prescott, 2004; Goodwin & Gotlib, 2003), as well as agreement in the literature that neuroticism predicts VMIP susceptibility (Hill, 1985; Blackburn, Cameron & Deary, 1990; Schreindorfer, 2002).

H5: There will be a direct relation between negative attributional style and susceptibility to the negative VMIP.

Rationale: Relationships between pessimism/negative attributional style and naturally occurring depression (Parker, Wilhelm & Asghari, 1998; Hartlage, Arduino & Alloy, 1998; Abramson et al., 2000).

H6: There will be a direct relation between self critical perfectionist traits and susceptibility to the negative VMIP.

Rationale: Recent evidence showing that self critical perfectionism is correlated with naturally occurring negative affect (Powers, Zuroff & Topciu, 2004; Dunkley, Zuroff & Blankstein, 2003)

H7: There will be a direct relation between ruminative cognitive style and susceptibility to the negative VMIP.

Rationale: Rumination has been linked to naturally occurring depression and depressive symptoms (Alloy et al., 2000; Robinson & Alloy, 2003; Just & Alloy, 1997; Kuehner & Webber, 1999).

H8. There will be a direct relation between high scores on the Silencing the Self Scale and susceptibility to the negative VMIP.

Rationale: Theory and research suggesting that silencing of the self may explain the emergence of clinical depression (Jack, 1991; Jack & Dill, 1992; Stoppard, 2000)

Relative contribution of individual characteristics.

H9. The relative contributions of the above characteristics to vulnerability to induced depressed mood will be examined using a regression analysis. No specific hypotheses are proposed for this analysis.

Chapter II

Method

*Participants**Participant Numbers and Characteristics*

One hundred and eighty-eight (149 women; 39 men) were included in the original data set used in this study. All participants were students attending the University of Windsor between June and August 2005. All survey sets that were missing 5% or more of responses were deleted from the data set. In total 10 participants were deleted from the original data set, resulting in the final data set consisting of 178 participants (145 women, 33 men) with an average age of 23.7 years ($SD=6.616$; ranging from 18 to 50 years of age). For all participants with less than 5% of their total data missing, missing values were replaced using the EM missing value replacement method. The EM method uses an iterative process to estimate the means, covariance matrix, and correlation of quantitative variables in order to replace missing values. In the final data set, 28 participants (16.3% of the sample) reported having been previously diagnosed with depression, while 149 reported never having been diagnosed with depression (one participant did not respond). The level of previous diagnosis reported in this study is four times higher than the Canadian average of depression diagnosis (Patten & Lee, 2004), but it may still be an underestimate of the sample's previous diagnoses, given typical underreporting of mental health diagnosis due to stigma (Halter, 2004). This is four times higher than the average rate of depression diagnoses reported for the general Canadian population.

Recruitment Methods

All participants were recruited through the University of Windsor Psychology Participant pool. Participants were either contacted by phone, or signed up for participation via participant pool sign up sheets. Every participant was awarded 3 bonus points in the courses of their choice as thanks for their time.

*Measures**Locus of control.*

Levenson's Locus of Control Scale (LMLC; Levenson, 1974) was used to measure locus of control. The LMLC consists of 24 items and uses a six point Likert scale response format. This scale yields three subscale scores; Internal Control, Powerful Others and Chance, as well as an overall rating of locus of control, scores on which can range from 0 (internal locus of control) to 124 (external locus of control). Chance measures the belief that the world is unordered, Powerful Others measures the belief that powerful others are in control of the world, and Internality measures the belief that the participant is (or is not) in control of the world. Test-retest reliability for the LMLC quoted as .66, .62, and .73, for the three subscales respectively over 7 weeks (Levenson, 1981). The LMLC is widely used to measure locus of control (Leone & Burns, 2000). High convergent validity has also been found between the LMLC and other popular measures of locus of control (Goodman & Waters, 1987). In addition, the LMLC has been found to be more factorially stable than Rotter's Internality-Externality Scale (ROTC), while still having reliability and validity comparable to the ROTC (Blau, 1984).

Emotional intelligence.

Emotional intelligence was measured by the Emotional Intelligence Scale (EIS; Schutte et al., 1998), a 33 item measure with a five point Likert scale response format. Internal consistencies of .87 and test-retest reliability of .78 have been reported for the EIS (Schutte et al., 1998). Predictive validity includes longitudinal prediction of academic success ($r=.67$ Schutte, et al., 1998) as well as correlations between high levels of EQ and less alexithymia, greater attention to feelings, greater clarity of feeling, more mood repair and less depression (Schutte et al., 1998). Convergent validity with other EQ scales has also been observed (Tapia & Marsh, 2003).

Neuroticism.

Neuroticism was measured using the N subscale of the Eysenck Personality Questionnaire-Revised Short Scale (EPQ-RSS; Eysenck, Eysenk & Barrett, 1985). This subscale is embedded in a larger scale of 48 items and consists of 12 items scored using a yes or no response format. Previous researchers have administered solely this subscale to measure neuroticism (Kendler, Kuhn & Prescott, 2004). Test re-test reliabilities of .84 and .80 and internal consistencies of .88 and .85 have been reported for men and women respectively (Eysenck, Eysenck & Barrett, 1985). Research suggests that EPQ-R Neuroticism scale compares favourably to similar personality questionnaires in terms of internal consistency and comparable scale correlation (Angleitner, John & Lohr, 1986) and that the N scale has the most favourable psychometric properties of the subscales of the EPQ-R (Eysenk & Eysenck, 1994; Caruso et al., 2001).

Pessimism/Negative Attributional Style

A pessimistic style of interpreting events was assessed using the Attributional Style Questionnaire (ASQ; Peterson & Villanova, 1988). The ASQ asks respondents to provide ratings across scales assessing for internality, stability and globality. Participants are presented with 24 hypothetical bad events involving themselves. For each event, participants are asked to imagine the event happening to them. They then describe the one major cause of the event and rate it in terms of internality versus externality, stability versus instability, and globality versus specificity. Using this scale, individuals who believe that bad things (i.e. negative events) happen to them due to internal, stable and global factors are considered to have a pessimistic explanatory style. The EASQ measures responses to 24 negative events (it does not measure responses to positive events). Internal consistencies (Cronbach's alpha) of .66 for internal, .85 for stability and .88 for globality have been reported (Peterson & Villanova, 1988).

Self critical perfectionism.

Self critical perfectionism was measured using the method described by D. Dunkley (personal communication, May 2, 2005), who advocates assessing self critical perfectionism using the Depressive Experiences Questionnaire (DEQ; Blatt et al., 1976) self-criticism subscale. The DEQ consists of 66 items describing experiences frequently reported by depressed individuals (e.g., "There is a considerable difference between how I am now and how I would like to be"). Internal consistency of .75 (Zuroff et al., 1990) and test retest reliabilities ranging from .68 to .83 (Zuroff et al., 1983) have been reported for the self-criticism subscale of the DEQ. Similarities in scores and factor loading between men and women respondents has led to the general recommendation to score

both men and women using the original norms obtained for women (Blatt et al., 1976; Zuroff et al., 1990).

Rumination.

Rumination will was measured using the Rumination Response subscale (RRS) of the Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991). The RRS has been used by itself by previous researchers (e.g. Robinson & Alloy, 2003; Hankin et al., 2005). The RRS consists of 22 items describing self-focused, symptom focused, or consequence focused reactions to mood and uses a four point Likert scale ranging from "almost never" to "almost always". The RRS has been shown to predict depression (Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991). Internal consistencies (Cronbach's alpha) of .89 (Nolen-Hoeksema & Morrow, 1991), coefficient alphas of .92 (Hankin et al., 2005) and test-retest consistencies of .80 (Nolen-Hoeksema, Parker & Larson, 1994) and .67 (Treynor, Gonzalez & Nolen-Hoeksema, 2003) have been reported for the RRS.

Silencing the self.

This was measured by the Silencing the Self Scale (STSS; Jack & Dill, 1992). The STSS is designed to measure Jack's construct of relational causes for depression in women (Jack, 1991). The STSS consists of 31 items; five items are reversed to control for response set acquiescence. Possible scores range from 31 to 155. Four subscales include externalized self perception (items 6,7,23,27,28,31); care as self-sacrifice (items 1,3,4,9,10,11,22, 29); silencing the self (items 2,8,14,15,18,20,24,26,30); and divided self (items 5,13,16,17,19,21,25). Internal consistencies range from .86 to .94 (Jack & Dill, 1992). Test-retest validity has been reported to be in a similar range. Results supporting construct validity include significant correlations between STSS score and mild and

moderate depression, with participants with higher STSS scores reporting higher scores on depressive symptom measures (Jack & Dill, 1992; Stoppard, 2000). In addition, women with a history of sexual/physical abuse have higher STSS scores than women without a history of abuse (Stoppard, 2000).

Mood.

Mood was measured using the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS consists of 20 adjectives (10 positive, 10 negative) and has been designed to measure positive and negative affect. Daily ratings have been found to be reliable and valid measures of these two distinct dimensions of affect (Watson, Clark & Tellegen, 1988; Goff, 1999). Goff (1999) found test re-test reliabilities (coefficient alphas) for each of the 7 days for his study, and the average reliabilities over 7 days were .89 for Positive Affect and .83 for Negative Affect. Internal consistencies for PA and NA were .73 and .70 respectively. In addition, Goff found that BDI scores were consistently correlated with NA and PA ($r=.55$ and $r=-.32$ respectively). “Various checklist procedures such as the MAACL (Zuckerman & Lubin, 1965) have been used in assessing depressed and anxious mood. The PANAS has two advantages over such measures. First, the PANAS is a scaled measure rather than a checklist measure, allowing for a more sensitive measure of affect change. Second, the PANAS has better psychometric properties than other multi-affect measures” (Goff, 1999, p. 60).

Velten mood induction procedure.

Research by Schare and Lisman (1984) indicates that reading the full set Velten statements selected by Scheier and Carver (1977) has a greater impact than reading a reduced number of statements (25). As such, the VMIP statements selected by Scheier

and Carver from the original 60 VMIP statements were used. Scheier and Carver (1977) modified the original Velten statements to remove references to events that were “no longer relevant to contemporary society” (Kenealy, 1986, p. 319), for example, references to the Vietnam War (Finegan & Seligman, 1995). Each statement will be typed on a separate lineless index card (Scheier & Carver, 1977).

The specific VMIP procedure used was standard, as suggested by a number of researchers (Frost, Graf & Becker, 1979; Nagata & Trierweiler, 1988; Scheier & Carver, 1977; Finegan & Seligman, 1995, Velten, 1968). The following instructions were written on the first index card:

"These statements are designed to make you feel depressed. Read each of the following statements out loud to yourself. As you look at each statement, focus your observation only on that one. As you are reading each statement, try to feel the mood suggested by the statement. Please use the signals from the tape recorder to pace yourself. Each time you hear a tone, progress to a new card".

The cards were placed face down in front of the participant. Upon giving the above instructions, the experimenter pressed play on the audio tape. The audiotape consisted of a series of clicks, each spaced 15 seconds apart. Asking the participants to read each card at 15 second intervals is an arbitrary decision. Other research has used 10 second intervals (Scheier & Carver, 1977), asked participants to read all of the statements at their own pace for a total of seven minutes (Finegan & Seligman, 1995), or had the experimenter read the statements out loud to the participant (Hill, 1985). Based on meta-analysis results (Westerman, Spies, Stahl & Hesse, 1996) the most common procedure appears to be asking participants to read instructions typed on lineless index cards, and

then having participants read the statements out loud to themselves at 15 second intervals. Without evidence speaking to the most efficacious procedure, this study used the most common procedure.

Procedures

Participants were recruited from the University of Windsor participant pool. All participants experienced the same procedure. First, participants were given a consent form and letter of information. The consent form provided an option allowing participants to give the researcher permission to contact them for follow-up studies in 2 to 3 years time. This option allowed participants to write their participant identification number on the consent form, along with their names, which will be kept in a secure location. No other identifying information was associated with participant numbers. Upon signing the consent form, participants were given a package including the following: questions of demographic information including age, gender, and previous diagnosis of depression; the Positive and Negative Affect Scale (PANAS, Watson, Clark, & Tellegen, 1988) to measure mood, and the following questionnaires in random order:

Levenson's Locus of Control Scale (1974)

The Emotional Intelligence Scale (Schutte et al., 1998)

The Eysenk Personality Questionnaire-Revised Short Form (Eysenck, Eysenck & Barrett, 1985)

The Expanded Attributional Style Questionnaire (Peterson & Villanova, 1988)

Depressive Experiences Questionnaire (Blatt et al., 1976)

Rumination Response Subscale of the Response Style Questionnaire (Nolen-Hoeksema & Morrow, 1991)

The Silencing the Self Scale (Jack & Dill, 1992)

It took approximately 60 minutes to complete these questionnaires. Then participants were moved to a separate room where they were asked to take a break (have a drink of water, go to the bathroom, relax etc.) for 10 minutes. Finally, participants were taken into a separate room where they were asked to read 60 negative VMIP statements, using the procedure described above, which took exactly 15.75 minutes. Timing was regulated by playing beeps at 15 second intervals. Following this negative mood induction, participants were asked to complete a second PANAS. Moods induced by negative VMIP have been demonstrated to last approximately 10 minutes, and it has been recommended that participants in the negative condition will be asked to read a subset of elation statements in order to remove any remaining negative mood (Frost & Green, 1982). Accordingly, participants were then given 60 positive VMIP statements to read in order to address any ill effects of the negative mood induction; this took approximately 15 minutes. Participants were then debriefed and provided with contact information for the researcher as well as community mental health resources.

Chapter III

RESULTS

First, analyses were conducted to determine if, on average, the mood induction was successful. Success was defined as a significant decrease, following the negative VMIP, in the Happy and/or Sad mood scores on the PANAS. Responses to the Sad PANAS words were reverse scored, so that a higher score on the Happy PANAS scale indicated a less happy mood, and a higher score on the Sad PANAS scale indicated a more sad mood. Preliminary analyses explored the relationship between change in Happy and Sad PANAS scores, the effect of previous diagnosis of depression on responsiveness to negative mood induction, the relationship between participant age and responsiveness to negative mood induction, and the intercorrelations among the personality characteristics. After reviewing the results of the preliminary analyses, the main analyses will be discussed in the order of the hypotheses. The first main analysis examined any gender differences in change in mood scores pre- and post- negative mood induction. The second main analysis examined whether or not each of the personality traits measured was correlated with change in the Happy or Sad mood scores. Finally, the personality traits found to be significantly correlated to change in either the Happy or Sad mood scores were entered into one of two linear regressions (one for change in Happy mood and one for change in Sad mood) to determine their relative contribution, if any, to change in Happy and Sad mood scores.

Success of the Mood Induction

A paired samples t-test compared Sad PANAS scores before ($M=45.44$, $SD=5.33$) and after ($M=37.82$, $SD=7.92$) VMIP and Happy PANAS scores before ($M=30.46$,

SD=6.78) and after ($M=17.42$, $SD=7.08$) VMIP. Results indicate that participants were significantly sadder after experiencing the negative VMIP condition, $t(177) = 12.95$, $p < .01$, and were significantly less happy after experiencing the negative VMIP condition, $t(177) = 26.93$, $p > .01$. These results indicate that the negative VMIP had a large effect ($d = -1.12$) on change in happy moods, and a large effect ($d = 1.88$) on change in sad moods.

Only one participant reported being happier after the negative VMIP, all other participants reported experiencing no change in Happy mood (3 participants) or experiencing a decrease in happy mood to various degrees (174 participants). Nine participants reported being less sad after the negative VMIP, all other participants reported experiencing no change in Sad mood (44 participants) or an increase in sad mood to various degrees (125 participants).

Relationship Between Changes in Happy and Sad Mood Scores

As one would expect, change in the Happy PANAS score was directly related to change in the Sad PANAS score, $r = .322$, $p < .01$. However, this correlation was unexpectedly low; changes in the Happy and Sad PANAS scores shared only 10.3% of their variance.

Exploratory Findings: Previous Depression Diagnosis

An independent samples t-test was conducted to compare responsiveness to the negative VMIP between participants who had and had not been previously diagnosed with depression. Results indicated that there were no significant differences between individuals with and without a previous diagnosis in change in either Sad ($t(175) = .387$, $p > .05$) or Happy ($t(175) = -.534$, $p > .05$) PANAS mood scores.

Exploratory Findings: Age

An examination of the relationship between participant age and responsiveness to induced depressed mood was conducted using a bivariate correlation. Results indicate that age was significantly negatively correlated with change in Sad PANAS mood scores ($r = -.255, p < .01$), meaning that younger participants were more susceptible to the negative VMIP than older participants (see Table 1). Interestingly, age was not significantly correlated with change in Happy PANAS mood scores ($r = -.079, p > .05$).

In order to explore these findings further, age was correlated with the personality characteristics measured in the study (see Table 2). Age was significantly negatively correlated with neuroticism ($r = -.220, p < .01$), self critical perfectionism ($r = -.172, p < .05$) and rumination ($r = -.171, p < .05$). Age was not correlated with any other personality characteristic measured.

Correlations Among Personality Characteristics

In order to determine whether or not colinearity of personality characteristics would be an issue in the regression equation, the correlations between the personality characteristics were examined. Results indicate that there were numerous correlations between the personality characteristics measured (see Table 2).

Hypothesis 1: Gender.

Hypothesis 1 predicted that women would be more responsive to the negative Velten mood induction than men. This prediction was not confirmed by the data. Results

Table 1.

Correlations Between Change in Mood Scores and Personality Characteristics and Age

<i>Mood</i>	<i>Characteristic</i>	<i>R</i>
Change in HAPPY mood (from PANAS)	Emotional Intelligence	.235**
	Locus of Control	-.159*
	Silencing the Self Score	-.162*
	Neuroticism	-.005
	Self Critical Perfectionism	-.066
	Negative Attribution Scale	-.019
	Rumination	-.038
	Age	-.079
Change in SAD mood (from PANAS)	Emotional Intelligence	.045
	Locus of Control	.019
	Silencing the Self Score	.067
	Neuroticism	.195**
	Self Critical Perfectionism	.107
	Negative Attribution Scale	.051
	Rumination	.088
	Age	-.255**

Note. ** $p < .01$, * $p < .05$ (2 tailed)

Table 2.

Correlations Among Personality Characteristics and Correlations Between Personality Characteristics and Age

	<i>N</i>	<i>EQ</i>	<i>LOC</i>	<i>SSS</i>	<i>R</i>	<i>SCP</i>	<i>NAS</i>
Neuroticism (N)							
Emotional Intelligence (EQ)	-.460**						
Locus of Control (LOC)	.401**	-.485**					
Silencing the Self Scale (SSS)	.525**	-.484**	.602**				
Rumination (R)	.644**	-.282**	.416**	.543**			
Self Critical Perfectionism (SCP)	.727**	-.518**	.458**	.629**	.641**		
Negative Attributional Scale (NAS)	.346**	-.248**	.304**	.361**	.332**	.362**	
Age	-.220**	.133	-.016	-.064	-.171*	-.172*	-.053

Note. ** $p < .01$, * $p < .05$ (2 tailed)

from two independent samples t-test, with gender as the independent variable and change in Happy and Sad mood scores as the dependent variables, indicate that there are no significant differences between men ($M=11.75$, $SD=6.19$) and women ($M=13.26$, $SD=6.47$) in responsiveness to the negative VMIP with respect to changes in Happy mood, $t(176)=1.219$, $p>.05$ and no significant differences between men ($M=-6.39$, $SD=7.06$) and women ($M=-7.89$, $SD=8.01$) in responsiveness to the negative VMIP with respect to changes in Sad mood, $t(176)=0.989$, $p>.05$ (see Table 3).

Hypotheses 2-8: Correlations Between Individual Characteristics and Change in Mood.

Hypotheses 2-8 predicted that each of the individual personality characteristics measured would be correlated with responsiveness to the negative VMIP. More specifically, it was predicted that external locus of control, silencing the self, rumination, self critical perfectionism, neuroticism, and negative attribution style would be directly related to responsiveness to the negative VMIP, whereas emotional intelligence would be inversely related to responsiveness to the negative VMIP. For the purposes of this study, a positive score on the change in Happy mood variable constitutes responsiveness to negative VMIP (mood became less happy after the VMIP), and a positive score on the change in Sad mood variable constitutes responsiveness to the negative VMIP (mood became more sad after the VMIP). Scores of zero on both change in happy and sad mood variables indicate that there was no change in mood rating before and after mood induction. Results of these correlational analyses are presented in Table 1.

Hypothesis 2: Locus of Control.

Results from a bivariate correlation indicate that Locus of Control was inversely related to change in Happy PANAS mood score, $r = -.159$, $p<.05$. Contrary to the

Table 3.

Gender Differences in Responsiveness to the Negative Velten Mood Induction Procedure

	<i>t</i>	<i>df</i>	<i>p</i> (2-tailed)
Change in HAPPY PANAS mood scores Time1-Time 2	1.219	176	.224
Change in SAD PANAS mood scores Time 1-Time2	-.989	176	.324

predicted relationship, people with a more internal locus of control (lower locus of control scores) were more responsive to the negative VMIP (became less happy) than were people with a more external locus of control (higher locus of control scores). Locus of Control was not significantly correlated with change in Sad PANAS mood scores, $r = .019, p > .05$.

Hypothesis 3: Emotional Intelligence.

In contrast to the predicted relationship, emotional intelligence was significantly positively correlated with Change in Happy mood score ($r = .235, p < .01$); as emotional intelligence increased, responsiveness to the VMIP (decreased happiness) increased. Emotional intelligence was not significantly correlated with change in Sad PANAS mood scores, $r = .045, p > .05$.

Hypothesis 4: Neuroticism.

Neuroticism was not significantly correlated with change in Happy PANAS mood scores ($r = -.005, p > .05$). However, neuroticism was significantly positively correlated with change in Sad PANAS mood scores, indicating the predicted result; the more neurotic participants were, the more likely they were to be responsive to (become sadder after experiencing) the negative VMIP. These results provide partial support for the hypothesis that there would be a direct relationship between neuroticism and responsiveness to the negative VMIP.

Hypothesis 5: Negative Attributional Style.

Negative attributional style was not significantly correlated with change in Happy ($r = -.019, p > .05$) or Sad ($r = .051, p > .05$) PANAS mood scores, leaving the hypothesis

that there would be a direct relation between negative attributional style and responsiveness to the negative VMIP unconfirmed.

Hypothesis 6: Self Critical Perfectionism.

Self critical perfectionism was not significantly correlated with change in Happy ($r = -.066, p > .05$) or Sad ($r = .107, p > .05$) PANAS mood scores, leaving the hypothesis that there would be a direct relation between self critical perfectionism and responsiveness to the negative VMIP unconfirmed.

Hypothesis 7: Rumination.

Rumination was not significantly correlated with change in Happy ($r = -.038, p > .05$) or Sad ($r = .088, p > .05$) PANAS mood scores, leaving the hypothesis that there would be a direct relation between rumination and responsiveness to the negative VMIP unconfirmed.

Hypothesis 8: Silencing the Self Score.

Results from a bivariate correlation indicate that the Silencing the Self score was significantly negatively correlated with the change in the Happy mood score, $r = -.162, p < .05$. The negative correlation between the Silencing the Self score and change in the Happy mood score also indicates an unexpected relationship; the less people tended to silence themselves, the more responsive they were to the negative VMIP (the less happy they became). The Silencing the Self score was not significantly correlated with change in the Sad PANAS mood score, $r = .067, p > .05$.

Hypothesis 9: The Relative Contribution of Each Personality Characteristic

In order to examine the relative contribution of each personality characteristic to the prediction of change in happy and sad moods, two separate linear regressions (using a

stepwise entry method) were conducted. The first regression equation examined the relative contribution to change in the Happy PANAS mood score of each of the personality characteristics found to be significantly correlated with the change in this score (locus of control, emotional intelligence and silencing of the self) (see Table 4). Collinearity statistics were first examined to determine if collinearity was an issue in the regression. Results indicated that collinearity was at acceptable levels (collinearity for locus of control = .765, Silencing the Self = .766). The first step and model of the regression was significant, $F(1, 177) = 10.27, p < .05$. Of the personality characteristics measured, only Emotional Intelligence was predictive of change in Happy mood scores, accounting for 5.5% of the variance.

The second regression equation examined the relative contribution of the variables found to be correlated with change in the Sad PANAS mood score (neuroticism and age) in predicting change in this score (see Table 5). Collinearity statistics were first examined to determine if collinearity was an issue in the regression. Results indicated that collinearity was at acceptable levels (collinearity for neuroticism = .951). The first step and model of the regression was significant, $F(1, 177) = 12.24, p < .01$, with age predicting 6.5% of the variance. The second step and model of the regression was also significant, $F(2, 178) = 8.17, p < .01$, with the addition of neuroticism predicting 2% of the variance. As such, age was a better predictor of responsiveness to the negative VMIP, as reflected by an increase in sad mood, than any of the personality characteristics measured.

Table 4.

Linear Regression Performed to Examine Which Personality Characteristics Predict Change in Happy PANAS Mood Scores

<i>Predictors</i>	<i>Model 1</i>	
	B	SE
EQ	.235*	0.036
R^2	.055	

Note. * $p < .05$ (2 tailed)

Table 5.

Linear Regression Performed to Examine which Personality Characteristics (and Age) Predict Change in Sad PANAS Mood Scores

<i>Predictors</i>	<i>Model 1</i>		<i>Model 2</i>		<i>Model 3</i>	
	β	<i>SE</i>	β	<i>SE</i>	B	<i>SE</i>
Age	-.255**	.087	-.223**	.088	-.225**	.087
Neuroticism			.146*	.182	.226**	.201
R^2	.065		.085		.109	

Note. * $p < .05$, ** $p < .01$ (2-tailed)

Chapter IV

DISCUSSION

The present study investigated the relationship between a number of personality characteristics and responsiveness to induced depressed mood. None of the study's hypotheses were completely supported, and only one hypothesis found partial support. However, the study has yielded some interesting findings. Of particular note are the differences found between decreased happiness and increased sadness and the relevance of emotion regulation in understanding these differences. The study's specific results, and their relationship to the literature, will be discussed first. This will be followed by an examination of the broader implications of the findings in the context of the theoretical and empirical literature. Finally, limitations of the study and suggestions for future research will be presented.

*Previous Depression Diagnoses.**Previous Depression Diagnoses.*

Results from this study do not indicate that responsiveness to induced depressed mood is affected by a previous diagnosis of depression. This result is contrary to what the scar hypothesis would predict. The scar hypothesis posits that the experience of depression alters a person's personality (Shahar et al., 2004). Furthermore, researchers have identified physiological changes associated with experiencing depression, including low folic acid levels (Sachdev et al., 2005), reduced heart-rate variance (associated with increased risk for heart disease) (Stewart, Yusim & Desan, 2005) and reduction in hippocampal volume (particularly in the right hippocampus) (Videbech & Ravnkilde, 2004). Given the physiological and personality changes that seem to result from

experiencing clinical depression, it is surprising to find that there are no differences in susceptibility to induced depressed mood with respect to previous depression diagnosis. This null finding, however, should be interpreted with caution. Research indicates that, often due to the stigma associated with a diagnosis of a mental illness such as depression, it is common for people to underreport being so diagnosed (Halter, 2004).

Age.

Age predicted increased sadness in response to induced depressed mood above and beyond any of the personality characteristics measured in the present study. The older a person was, the less sad she or he became after experiencing the negative mood induction. In other words, younger participants were more susceptible to the negative mood induction than older participants -- they became sadder. Age was also negatively correlated with neuroticism, rumination, and self critical perfectionism, a finding consistent with previous research. Results from a study of the Big Five personality traits (Neuroticism, Extraversion, Agreeableness, Openness, Conscientiousness) and aging among Italian adults between 20 and 80 years of age indicates that men “decline steadily in emotional stability [increase in neuroticism] across ages, whereas females steadily gain emotional stability [decrease in neuroticism] from young adulthood to mid-50s, after which their emotional stability declines” (Caprara, Caprara & Steca, 2003, p. 144). Thus the results from the present study may be indicative of a lesser degree of emotional stability in the predominant developmental stage of the participants. This interpretation also corresponds with results indicating that neuroticism was predictive of responsiveness to increased sad mood after the negative mood induction and negatively correlated with age. Similar findings, that ‘positive’ personality characteristics increase with age while

‘negative’ personality characteristics decrease, have been reported in the literature. A retrospective study of 259 participants found that “Participants reported higher levels of identity certainty [and] confident power” at age 60, then at age 40, and then at age 20 (Miner-Rubino, Winter & Stewart, 2004, p. 1599).

The present investigation’s age-related findings are particularly striking given the narrow age range of the participants -- 82% of the participants were between 18 and 25 years of age, with only 23 participants above 30 years of age. As a result of this narrow age range, these results may be indicative of responsiveness to induced depressed mood at a specific developmental stage, as opposed to across the lifespan. Previous research examining age related changes in the clinical expression of psychiatric symptoms highlighted young adulthood (age ranging from 18 to 25 years of age) as a time when people are potentially prone to developing delusional ideation (Verdoux et al., 1998). Their study, based on a sample of 444 participants without psychiatric disorders, found an “age-related decrease in the likelihood to report delusional ideas...[with] younger subjects scoring higher on most dimensions of delusional ideation, such as ‘persecution’, ‘thought disturbance’, ‘grandiosity’ and ‘paranormal beliefs’” (Verdoux et al., 1998, p. 247). This suggests that individuals 18 to 25 years of age are more vulnerable to psychiatric symptomatology than are older adults, consistent with the findings of the present study that participants’ tendency to become sadder following the negative mood induction was directly related to their age.

Gender.

The present study found no gender differences in responsiveness to the induced depressed mood. A number of studies have found gender to be unrelated to VMIP

responsiveness (Blackburn, Cameron & Deary, 1990; Buchwald et al., 1981; Cairns & Norton, 1988; Lewis & Harder, 1988). These results are also consistent with longitudinal research by Leadbeater and Barker (2004), which found no difference between risk factors for depressive vulnerability between adolescent boys and girls. Also consistent with these results is information from Westerman et al.'s (1996) meta-analysis of the VMIP. They found that gender did not contribute to VMIP effect size. This lack of gender differences is inconsistent with the difference in diagnostic rates among men and women, where women are two to three times more likely to be diagnosed with depression than men (Culbertson, 1997; Sachs-Ericsson & Ciarlo, 2000). The present results indicate that men and women are equally vulnerable to induced depressed mood and, as such, one would expect them to be equally vulnerable to naturally occurring depressed mood. Other factors, such as artefactual and psychosocial explanations (Kuehner, 2003; Jack, 1991) might explain these epidemiological differences more accurately than inherent gender differences in responsiveness to sad moods. However, the apparent contradiction between the lack of gender differences in responsiveness to induced depressed mood and the clear gender differences in diagnosed depression might suggest that induced depressed mood does not parallel naturally occurring depressed mood, at least for university aged people.

Locus of Control.

Despite evidence in the literature linking external locus of control to naturally occurring depression (Lamanna, 2001; Benassi, Sweeny & Dufour, 1998; Jaswal & Dewan, 1997), results indicate that locus of control was not correlated with participants' increase in sad mood after the negative mood induction. Previous research has also found

locus of control to be unrelated to VMIP responsiveness (Rexford & Wierzbicki, 2001; Lewis & Harder, 1988; Cairns & Norton, 1988). However, the present study found that individuals with a more internal locus of control exhibited a greater decrease in happiness in response to the negative mood induction than individuals with a more external locus of control. This is the opposite of the relationship reported between external locus of control and naturally occurring depression.

The finding relating internal locus of control to decrease in happy mood after negative mood induction is new to the literature. It is, however, consistent with the construct of locus of control, given that individuals with an external locus of control are described as believing that events are outside of their control (Clarke, 2004). The nature of the VMIP task requires individuals to attempt to control their emotions, and it seems as though individuals who do not believe they are capable of controlling events were not as effective at becoming less happy. Because the mood induction task is one in which people are asked to effect change in their emotions, this finding might reflect the intersection between efficacy (internal locus of control) and ability to effect emotional change. The decrease in happy mood following negative mood induction might reflect not a susceptibility to external influence, but rather an ability to consciously influence one's emotional experience.

Emotional intelligence.

Emotional intelligence was found to be directly related to change in Happy PANAS scores -- happiness in individuals with high emotional intelligence decreased more following negative mood induction than that in individuals with low emotional intelligence. Emotional intelligence predicted 5.5% of the variance in change in Happy

PANAS scores. Given that individuals with high emotional intelligence are postulated to be better able to monitor and control their emotions than individuals with low emotional intelligence, it is theoretically consistent that individuals with higher emotional intelligence instructed to feel sad were better able to do so than individuals with lower emotional intelligence. This finding is consistent with other literature (Petrides & Furnham, 2003) which concluded that high emotional intelligence allows people better control of their emotions, thus making participants better able to manipulate their emotions in the desired (in this case, negative) direction. It appears that the ability to monitor and be aware of one's emotions plays a key role in responsiveness to induced depressed mood. This finding provides quantitative support for the emotional intelligence construct. This finding may further clarify why high emotional intelligence is a protective factor with respect to naturally occurring depression (Lamanna, 2001). High emotional intelligence would aid people in situations where they may want to avoid sliding into depression by enabling them to increase their happiness. Depression is, after all, more than feeling very sad; it is also the inability to feel happy.

Neuroticism.

The relationship between neuroticism and induced mood has been one of the few consistent findings in the mood induction research to date (Hill, 1985; Blackburn, Cameron & Dearly, 1990; Schreindorfer, 2002). The present study also found that neuroticism was positively correlated with change in Sad PANAS scores. Individuals high in neuroticism became sadder after negative mood induction than individuals low in neuroticism. The unique contribution to the literature provided by this aspect of the present study is the examination of the predictive usefulness of neuroticism to

responsiveness to induced mood. Neuroticism was not found to be related to a decrease in happy mood. Other literature has also found a difference between neuroticism and happy and sad moods. Larsen and Ketelaar (1991) found that “that neuroticism shows the strongest relationship to affect when negative affect is manipulated and measured. Extraversion, on the other hand, shows the strongest relationship to affect when positive affect is manipulated and measured.” (p. 136). This corroborates other studies which have found that extraversion correlates with and predicts levels of positive affect and neuroticism correlates with and predicts negative affect (Costa & McCrae, 1980 as cited in Larsen & Ketelarr, 1991). Thus research supports the finding that neuroticism is more related to sad than happy moods.

Pessimism/Negative Attributional Style, Self Critical Perfectionism and Rumination.

Despite literature linking naturally occurring depression vulnerability to pessimism (Abramson et al., 2000; Powers, 1997; Parker, Wilhelm & Asghari, 1998; Hartlage, Arduino & Allo, 1998; Kapçi & Cramer, 2000), self-critical perfectionism (Powers, Zuroff & Topciu, 2004; Dunkley, Zuroff & Blankstein, 2003; Cox et al., 2004) and rumination (Papageorgiou & Siegle, 2003; Alloy et al., 2000; Robinson & Alloy, 2003), the present study found no relationship between these characteristics and responsiveness to induced negative mood. The present study is the first to examine these characteristics with respect to VMIP responsiveness.

Silencing the Self.

The Silencing the Self scale provides a unique and empirically validated measure designed to examine women’s experience with depression from a relational theoretical model (Jack, 1996). Individuals who score highly on the Silencing the Self Scale are

described as “putting the needs of others first, censoring and repressing genuine emotion, and judging themselves from external standards” (Cramer & Thoms, 2003, p.526).

Results from the present study indicate that happiness scores did not decrease as much after negative mood induction for individuals high in silencing the self as it did for individuals low in silencing the self. Though this result was not predicted, it does support the ‘repressing genuine emotion’ aspect of the construct. Individuals high on silencing the self tend to deny their own emotions in favour of others (Jack, 1996). When asked to monitor and change their emotions, high silencing the self individuals were unable to do so as effectively as low silencing the self individuals. The low silencing the self style permits greater awareness of one’s emotions, a prerequisite to changing one’s emotions.

Change in Sad Mood Versus Change in Happy Mood

An unexpected finding that emerged from the present study is the difference between the characteristics related to increase in sad mood and those related to decrease in happy mood. To summarize, neuroticism and age predicted and were correlated with change in Sad PANAS score but not at all related to change in Happy PANAS score. Emotional intelligence, silencing the self and locus of control were all correlated with change in Happy PANAS score, but not at all related to change in Sad PANAS score. The correlation between change in Happy and Sad PANAS mood scores was also small; only 10.3% of the variance of each was shared between the two. Results indicating that happy and sad mood scores, as measured by the PANAS, are only weakly correlated have been called a “common finding” (Larsen, McGraw & Cacioppo, 2001, p. 686). For example, Larsen and Ketelaar (1991) found that the positive and negative affect scales correlated at .23, and that a positive mood induction (imagining they won the lottery)

affected positive but not negative affect (happy mood increased but sad mood did not decrease). These results support the notion that happy and sad moods are not on the same continuum (i.e. not the polar opposites of each other), but rather are separate, but related, mood states.

Traditionally, happy and sad moods have been conceptualized as diametric, existing at “opposite ends of a bipolar continuum” (Larsen, McGraw & Cacioppo, 2001, p. 685). This theoretical explanation of the relationship between happy and sad moods has been described by Russell and Carroll (1999) as mutually exclusive; “bipolarity says that when you are happy, you are not sad and that when you are sad, you are not happy” (p. 25). Recent investigations by Larsen, McGraw and Cacioppo (2001) have questioned this conceptualization of the relationship between happy and sad moods. They found that people are capable of feeling both happy and sad at the same time in a variety of emotionally complex situations (watching the movie “Life is Beautiful”, moving out of dormitories, and graduation day). Neurobiological evidence also supports the conceptualization of happy and sad moods as separable emotions. The amygdala has been associated with negative affect (LeDoux, 1995, as cited in Larsen McGraw & Cacioppo, 2001) and the mesolimbic dopaminergic pathway has been associated with positive affect (Hoebel, Rada, Mark & Pothos, 1999 as cited in Larsen McGraw & Cacioppo, 2001). Recent theoretical models, such as the Evaluative Space Model (ESM; Cacioppo, Gardner, & Berntson, 1999) have adapted to the evidence that happy and sad moods are separate but related emotions. The ESM postulates two separate and partially distinct aspects of emotion; “one attuned to nurturance and appetition (i.e., positivity), and the other attuned to threat and aversion (i.e., negativity)” (Larsen McGraw &

Cacioppo, 2001, p. 686). In this model, positive and negative emotions can be reciprocally activated (e.g., positive increases, negative decreases), but uncoupled activation (e.g., positive increases, negative does not change), or co-activation (e.g., positive and negative both increase) can also occur. Thus, positive and negative affect can be negatively correlated (as is predicted in diametric models) but can also be positively correlated or uncorrelated. The ESM model does predict that, “If the affect system evolved to guide behavior, however, we would expect co-activation to be unpleasant, unstable, and often short-lived.” (Larsen McGraw & Cacioppo 2001, p. 687). As such, the ESM model suggests that most emotional experience would feel, for example, either happy or sad in typical situations, but that complex emotional situations would reveal the underlying separate constructs of positive and negative moods.

Emotion Regulation.

The field of emotion regulation provides potential clarification for some of the findings of the present study. Emotion regulation is defined as a person’s ability to “tolerate, be aware of, put into words, and use emotions adaptively to regulate distress and to promote needs and goals” (Elliott et al., 2002, p. 32). Adaptive emotion regulation has been identified as necessary for adaptive functioning (Elliott et al., 2002), whereas maladaptive emotion regulation has been found to be associated with mental health problems (Gross & Munoz, 1995). Specifically, dysfunction of emotion regulation has been found to be significantly associated with depression among college students (Min’er & Dejun, 2001). With respect to age, research indicates that there is evidence for a shift towards a more healthy type of emotion regulation in people as they age (John & Gross, 2004). This may partially explain the present study’s finding that younger individuals

were more susceptible to the negative mood induction. Results from research by John and Gross (2004) and Kokkonen and Pulkkinen (2001) also indicate that neuroticism is correlated with maladaptive emotion regulation style. Again, this corresponds to the present research, which found that the more neurotic the participant, the sadder they became after experiencing the negative mood induction. With respect to emotional intelligence, adaptive forms of emotional regulation have been described as necessary precursors for developing emotional intelligence (Zeidner, et al., 2003; Arsenio, 2003). As such, individuals with high emotional intelligence would, by definition, practice adaptive emotional regulation strategies. Correspondingly, the present study's findings that individuals with higher emotional intelligence are more able to reduce their happiness in response to negative mood induction suggests that individuals with more successful emotion regulation strategies are better able to manipulate their emotions in desired directions.

Conclusions.

Results from this study have shown that different personality variables are related to change in happy mood than are related to change in sad mood. Change in happy mood is correlated with characteristics related to emotion regulation. Individuals with an internal locus of control, low levels of silencing the self, and high emotional intelligence are more able to reduce their happiness when asked to do so than are people with an external locus of control, high levels of silencing the self, and lower emotional intelligence. In short, such individuals are better at regulating their happy emotions than are their counterparts. Interestingly, changes in sad mood tell a different story. Participants high in neuroticism became sadder than their low neuroticism counterparts

following negative mood induction. In contrast to change in happy mood, change in sad mood appears to be related more to susceptibility to externally generated negative thoughts (as supplied by the VMIP). Research has shown that neuroticism is associated with maladaptive emotion regulation. As such, it is possible that highly neurotic participants became sadder as a result of associated maladaptive emotion regulation. Participants with adept emotion regulation (high emotional intelligence, internal locus of control, low silencing the self) allowed themselves to become less happy to comply with instructions, but did not allow the more threatening experience of becoming sadder to occur. In contrast, participants with less adept emotion regulation (high neuroticism) were vulnerable to the more threatening experience of increased sadness. These results support the view of sad and happy moods as separate and only partially related emotions, with different processes and personality characteristics contributing to each.

Limitations of the Study.

There are two important limitations to this study that must be considered. This study was conducted entirely using university students. Interpreting the results as applying to the broader population would be premature. In addition, the characteristics and moods measured in the present study were assessed entirely using self report measures. The limitations inherent in the use of self-report measures apply to this study.

Future Research Directions.

Two particular lines of investigation are suggested by the present investigation. Research is needed to examine the relationship of induced depressed mood to naturally occurring depressed mood and to clinical depression. The present investigation found no relation between a history of clinical depression and responsiveness to negative mood

induction, and that personality characteristics related to responsiveness to negative mood induction differed considerably from those reported in the literature as being related to naturally occurring depressed mood and clinical depression. How are we to interpret this? A second line of research suggested is predictive in nature. A longitudinal study is planned by the author to determine whether responsiveness to negative mood induction (with regard to both decrease in happiness and increase in sadness) predicts frequency or intensity of naturally occurring depressed mood or clinical depression later on in life. If individuals in the present study who tended in particular to become sadder in response to the negative VMIP (those high in neuroticism) should become more depressed (or are more likely to suffer depression) later in life, whereas individuals in the present study who tended in particular to become less happy in response to the negative VMIP (those high in emotional intelligence, those low in silencing the self, and those with an internal locus of control) should become less depressed (or less likely to suffer depression), then responsiveness to the VMIP would a clinically useful method of identifying at risk individuals.

Implications.

If the results of the present study are cross-validated in future investigations, they will have important implications for the prevention of naturally occurring depression. Individuals with an internal locus of control, low silencing the self, and high emotional intelligence were found to be able to regulate their emotions in order to manage their happy and sad moods in a directed and self protecting manner. Such individuals are receptive to the negative mood induction (they became less happy), yet were able to manage their emotions to avoid the more threatening experience of becoming sadder.

With respect to naturally occurring depressed mood, such individuals should be able to regulate their emotions to protect themselves against depressed moods when exposed to naturally occurring negative thoughts or experiences. Similarly, individuals high in neuroticism were found to be susceptible to the negative mood induction, becoming sadder in response to the depressing statements. Such individuals would be less successful at protecting themselves against sad moods when exposed to similar negative thoughts or experiences in the real world. This study has provided a theoretical explanation for empirical research that has found that internal locus of control (Lamanna, 2001; Benassi, Sweeney, & Dufour, 1988), high scores on the Silencing the Self scale (Jack, 1991; Jack & Dill, 1992; Stoppard, 2000) and high emotional intelligence (Lamanna, 2001; Ciarrochi et al., 2002) are protective factors with respect to naturally occurring depression and that high levels of neuroticism are predictive of naturally occurring depression (Watson, Gamez & Simms, 2005; Schmitz, Kugler & Rollnik, 2003; Kendler, Kuhn & Prescott, 2004; Goodwin & Gotlib, 2003).

Results of the present study, if cross-validated, will also have implications for the treatment of clinical depression. Treating a client's depressed mood is not *necessarily* enough to ensure a happy mood state, given that happiness and sadness are distinct dimensions, only partially related to one other. Furthermore, therapeutic assessment of personality characteristics must be informed about the fact that different personality characteristics are differentially related to positive and negative affect. In short, the present investigation provides material which may better inform both treatment and prevention strategies for clinical depression.

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Appendix A: Informed Consent/Consent Form



CONSENT TO PARTICIPATE IN RESEARCH

Title of Study: Individual Differences in Susceptibility to Induced Depressed Mood

You are asked to participate in a research study conducted by Laura Garcia-Browning at the University of Windsor. Results of this study will contribute to the researcher's M.A. thesis project. If you have any questions or concerns about the research, please feel free to contact Laura Garcia-Browning (garciab@uwindsor.ca) or Dr. Jim Porter (253-3000 ext. 7012, jporter@uwindsor.ca).

PURPOSE OF THE STUDY

This study is designed to examine personality characteristics which contribute to the experience of a depressed mood.

PROCEDURES

All procedures will be conducted in CHS room 181. If you volunteer to participate in this study, you will be asked to do the following: First, you will be asked to rate your current mood. Then you will be asked to fill out a number of questionnaires about yourself. Following this you will be asked to relax in a separate room for 10 minutes. Then you will be asked to read a series of statements designed to help you experience a sad or depressed mood. Then you will be asked to rate your mood again. Finally, you will be asked to read a series of statements designed to help you feel a happy or elated mood. The entire procedure should take approximately 2 hours to complete.

POTENTIAL RISKS AND DISCOMFORTS

This experiment involves experiencing a sad mood for approximately 10 minutes. This mood will be dissipated after 10 minutes, and every attempt will be made to help you leave the study feeling a more positive mood than when you began the study.

POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

Participating in this study will result in 3 bonus points on the course indicated by you. Your participation in this study will also help provide valuable information regarding potential prevention measures for feelings of sadness and depression.

PAYMENT FOR PARTICIPATION

Participants may be eligible for up to 3 bonus points in Psychology courses which permit bonus points.

CONFIDENTIALITY

Any information that is obtained in connection with this study that can be identified with you will remain confidential and will be disclosed only with your permission. Such information will be kept under lock and key for 3 years. No identifying information (such as your name or student number) will be attached to any of the data collected in this study. Your name will only be on this letter of consent. All data collected in this study (which will include survey results and mood ratings) will be kept under lock and key for 7 years, and then destroyed.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. An alternative task will be provided if you choose to withdraw from this study and still wish to be compensated for your time. You may also refuse to answer any questions you don't want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. You have the right to have your data removed from this study at any point during the experimental proceedings. Following the experimental proceedings your data cannot be removed from this study.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE SUBJECTS

If you are interested in obtaining a copy of the results once the study is complete, the results will be posted in the Windsor Research Ethics Board website at <http://athena.uwindsor.ca/reb> as of November 2005.

SUBSEQUENT USE OF DATA

If you give your consent, this data may be used in subsequent studies. In addition, the researchers ask for your permission to contact you at a future date (2-3 years from now) in order to ask you questions about your experiences with depression and/or depressed moods.

Do you give consent for the subsequent use of the data from this study? ☐ Yes ☐ No
 Do you give consent for the researchers to contact you in the future with regards to your experiences with depression and/or depressed moods? ☐ Yes ☐ No

If YES,

Please write your participant number here: _____

Please provide a phone number and email address that we can use to contact you in the future:

 Phone

 email

(All of the above information you have chosen to provide will remain confidential and will be destroyed in 3 years)

RIGHTS OF RESEARCH PARTICIPANTS

You may withdraw your consent at any time and discontinue participation without penalty. This study has been reviewed and received ethics clearance through the University of Windsor Research Ethics Board. If you have questions regarding your rights as a research subject, contact: Research Ethics Coordinator, University of Windsor, Windsor, Ontario, N9B 3P4; telephone: 519-253-3000, ext. 3916; e-mail: lbunn@uwindsor.ca.

SIGNATURE OF RESEARCH SUBJECT/LEGAL REPRESENTATIVE

I understand the information provided for the study Individual differences in susceptibility to induced depressed mood as described herein. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

 Name of Subject

 Signature of Subject

 Date

SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

 Signature of Investigator

 Date

Please keep one copy of this consent form for your own information and sign the other copy if you agree to participate in the study.

Appendix B: Debriefing Form



Individual Differences in Susceptibility to Induced Depressed Mood

Feelings of sadness are a central component of clinical depression, a devastating disorder which affects 3-4% of Canadians each year. Despite the prevalence and severity of this illness, little is known about individual characteristics which make people more vulnerable to experiencing feelings of unhappiness. A better understanding of the relationship between personality characteristics and depressed mood vulnerability can provide valuable information for both the treatment and prevention of Major Depression. The purpose of this study is to examine how various personality characteristics influence vulnerability to feeling a depressed mood.

Experiencing a sad mood is by definition an unpleasant experience, and your generosity and willingness to participate in this study are greatly appreciated. Your input will help contribute to the advancement of the field of depression research. If answering any of these questionnaires or experiencing this sad mood has led you to feel distressed and you would like to speak to someone about your thoughts, please contact one of the following:

Student counselling services: CAS Student Centre, Room 293, 253-3000 ext. 4616.
Teen Health Centre: 1585 Ouellette Avenue, 253-8481

If you are interested in obtaining a copy of the results once the study is complete, the results will be posted in the Windsor Research Ethics Board website at <http://athena.uwindsor.ca/reb> as of November 2005. If you are interested in this area of research, you may wish to read the following references:

Blackburn, I., Cameron, C., Deary, I. (1990). Individual Differences and Response to the Velten Mood Induction Procedure. *Personality and Individual Differences*, 11(7), 725-731.

Frost, R., Green, M. (1982). Velten Mood Induction Procedure Effects: Duration and Post-Experimental Removal. *Personality and Social Psychology Bulletin*, 8(2), 341-347.

Martin, M. (1990). On the Induction of Mood. *Clinical Psychology Review*, 10, 669-697.

If you have any complaints, concerns, or questions about this research, please feel free to contact Laura Garcia-Browning, Dr. Jim Porter (253-3000 ext. 7012, jporter@uwindsor.ca), or the Research Ethics Board at Windsor University (519-253-3000, ext. 3916, ethics@uwindsor.ca).

Thank you very much for participating!

VITA AUCTORIS

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